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SPECIAL ISSUE
XII MEETING
ARMED FORCES MEDICAL RESEARCH COMMITTEE

VOL. XXX JULY 1974 No. 3

ARMED FORCES MEDICAL JOURNAL (INDIA)

PUBLISHED QUARTERLY BY
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ARMED FORCES MEDICAL JOURNAL
(INDIA)

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EDITORIAL

The XII meeting of the AFMRC was held in Pune during February 1974, which was opened by Lt Gen Ved Parkash, PVSM, Director General Armed Forces Medical Services in his capacity as the chairman. His address was devoid of platitudes but full of sound rational advice on research in the Armed Forces.

Dr. A. K. Sundaram, Director Bio-medical Group, Bhabha Atomic Research Centre and Professor SAM G. P. Moses of the Government General Hospital and Madras Medical College, who were the guest speakers, spoke on "Nuclear Medicine—Present and Future Trends" and "Modern Concept of Early Diagnosis and its Value in the Clinical Approach and Management of Diabetes Mellitus". The subjects were covered very lucidly and their talks have been reproduced in this issue.

A conference of this nature should have wide publicity, especially in the interest of those officers, who were not able to attend. In order to achieve this purpos, a special issue of this Journal is being published. Our gratitude goes to the Commandant and staff of AFMC, who did the real work in planning and organising this meeting.

We are at present in the preliminary stage of planning for the XIII meeting to be held in Pune during February 1975, where our tradition for improvement and innovation will continue. Papers will be called for shortly on various aspects of actual work done in clinical medicine, research, development of new surgical techniques or any other subject where medical, dental and nursing officers have done original work. The criteria for selection of papers will in general be in terms of originality, methodology, scientific import and presentation. However, the rejection of a paper must not be regarded as a reflection on its special merit, since we have to consider diversity and balance in preparing a programme.

Demands for our Journal are being received from many European and Middle East Countries. It is also being abstracted by "Excerpta Medica", which is published from. Holland, has branches in USA, England, Italy, Japan and Argentina—this will no doubt encourage contributors to send their better articles to this Journal rather than publishing them elsewhere.

The Editorial Board have been making a concerted effort to continuously keep the professional standard of the Journal at par with other leading publications. The format is being changed and a new design of the cover page wlll be seen in the October issue. However, the success of this Journal depends on you and the articles you send us for publication. You are again reminded to follow all instructions given for contributors when forwarding your articles to the Editor.

A system of abstracting and book reviews has been introduced, but its success will depend on the response of the subscribers.

INTRODUCTION

Armed Forces Medical Research Committee which was recently reconstituted by the Government of India comprised of the following members :—

Lt Gen Ved Parkash, PVSM, DG, AFMS — Chairman

MEMBERS

1. Lt Gen P.V. Ramniah, PVSM, DMS (Army)
2. Rear Admiral J.N. Ghose, PVSM, MC, DMS (Navy)
3. Air Vice Marshal Ajit Nath, PVSM, DMS (Air)
4. Lt Gen A.K. Barat, PVSM, MVC, Chief Cons (S)
5. Maj Gen R.S. Hoon, AVSM, Senior Cons (M)
6. Maj Gen C.V. Pillai, PVSM, DDMS, Central Command
7. Maj Gen B. Mahadevan, AVSM, Commandant, AFMC, Pune
8. Maj Gen B.D.P. Rao, Senior Consultant (Pathology)
9. Air Cdre B. Bhatia, DRBS
10. Brig V. Rangaswamy, Vr C, DDMS II Corps
11. Brig S. Ganguly, DDMS IV Corps
12. Air Vice Marshal J.H.F. Manekshaw, AVSM, DMR, Member Secretary

BY INVITATION

Maj Gen D.B. Doctor, DDMS, Southern Command

NON-OFFICIAL MEMBERS

1. Dr. B.K. Anand, Professor & Head of Deptt. of Physiology, All India Institute of Medical Sciences, New Delhi*
2. Dr. Sujoy B. Roy, Professor & Head of Deptt. of Cardiology, All India Institute of Medical Sciences, New Delhi.
3. Lt Gen Inder Singh, Emeritus Scientist to the Armed Forces.
4. Dr. P.N. Gupta, Director, Virus Research Centre, Pune

---

* Now reassigned to W. H. O., New Delhi.

5. Dr. A.S. Paintal, Director, Patel Chest Institute, Delhi.
6. Dr. H.S. Bhatt, Professor of Surgery, Christian Medical College, Vellore-4

On 1 Apr. 73 there were 195 research projects on record with the DMR and during the financial year 1973-74.

(a) 24 were finalised.
(b) 14 were closed by the DG AFMS, since no progress had been achieved.
(c) 24 were recommended for closure by the XIth AFMRC in January, 1973.

leaving 133 current projects for consideration by the XIIth AFMRC in February, 1974, who recommended that 43 be closed immediately and the remaining 90 should be progressed.

142 new projects were presented to the XIIth AFMRC, who rejected 4 and accepted 98, with certain modifications.

The financial implications of these 186 projects amount to Rs. 10 lacs for the year 1974-75, which was approximately 6 lacs in 1973-74 and about 2 lacs on an average in previous years. The main cause of increase in the budget is due to the sudden activation of Naval and Dental Research, as also to a general impetus to research in the Armed Forces.

A certain amount of money has been kept in reserve by the DMR to cater for any contingency during the year.

AFMRC Forms 1 & 2 for new and current projects have been revised and are to be used with immediate effect. Specimen copies have been appended as appendices to the Administrative Conference Agenda and Minutes.

ARMED FORCES MEDICAL RESEARCH COMMITTEE MEETING

ADDRESS BY

Lt Gen Ved Prakash PVSM

26 Feb. 74

Ladies & Gentlemen,

I welcome the members of the AFMRC and all those who have come to attend the XII Meeting of this Committee. In Feb. 73, the membership of the non-official members of this Committee expired and the Raksha Mantri nominated a new panel comprising of :—

1. Dr. Sujoy B. Roy, Professor of Cardiology,
   All India Institute of Medical Sciences,
   New Delhi.
2. Dr. B.K. Anand, Adviser on Medical Education for South East Asia,
   W.H.O., New Delhi.
3. Dr. A.S. Gupta, Director,
   Patel Chest Institute, Delhi.
4. Dr. P.N. Gupta, Director,
   Virus Research Centre, Pune.
5. Dr. H.S. Bhatt, Professor of Surgery,
   Christian Medical College, Vellore.
6. Lt Gen Inder Singh, (Retd),
   Emeritus Scientist to the Armed Forces,
   New Delhi.

Dr. Sujoy B. Roy, Dr. B K. Anand, Dr. P.N. Gupta and Lt Gen Inder Singh have not been able to attend due to unforeseen circumstances. Dr. Anand has requested to be relieved from the membership of this Committee since he has been assigned to a new appointment in the World Health Organisation. However, I am pleased to have Dr.

A.S. Paintal and Dr, H.S. Bhatt with us today. I am also very happy to have with us Lt Gen PV Ramaniah, DMS (Army) and the other new Service members Maj Gen K.V. Pillai, DDMS, Central Command, Maj Gen D.B. Doctor, DDMS, Southern Command, Maj; Gen B.D.P. Rao, Senior Consultant (Pathology) and Air Vice Marshal J.H.F. Manekshaw, Director Medical Research and Brigadier V. Rangaswamy, DDMS II Corps who are participating in the meeting for the first time in their present appointments. I am sure that their vast experience in the field will be of immense value to us in our deliberations.

I am deeply conscious of the privileges of being the Chairman of this august body. I will, however, dispense with platitudes and come straight to issues which have been of some concern to me.

The financial position of the country does not require to be elaborated. I had advised the Expert Groups of the AFMRC to scrutinise all projects very carefully in order to avoid lavish or unnecessary expenditure and I would request this Committee to exercise the same vigilance within reasonable limits.

In the past R&D were not making any specific budget provision for the AFMRC, since the expenditure varied between 2–3 lacs, which they obtained from the budget of their other Establishments. However, in 1973 our budget was near 6 lacs, as such, the R&D requested us for a forecast budget for 1974 which has amounted to approximately 24 lacs plus 4.5 lacs equivalent in foreign exchange. But due to a directive from the Planning Commission, an increase of only 5% was permitted over the previous year's budget. It was, therefore, with considerable difficulty that I was able to make them agree to allocate Rupees 10 lacs for us this year, with perhaps another two lacs when the budget revision takes place in October. In the meanwhile I would ask you to ensure that your budget proposals are more realistic. I expect you to utilise the full amount when sanctioned, otherwise not only do we have to surrender the unspent sum but we also deprive some other organisation that may have used it more profitably.

The foreign exchange provisionally earmarked for us is about Rupees 50-55 thousand equivalent, but again let me emphasise that this depends on the amount allotted to R&D by the Government. It is quite possible this may not materialise as there is a negative balance of payment. Hence caution is to be exercised in its allocation to workers. Further, it should not be used up in just 2 or 3 projects.

Due the paucity of funds in relation to the demands, care has to be taken in sanctioning projects and scrutinising the budget requirement of each. Because of an element of uncertainty I would like the Committee to accord priorities to projects depending on their importance in case the budget is further reduced.

I have been gratified to see that in 1973-74 we closed 62 projects and the new ones increased from an average of 40 to 135. Hence I must remind you that it is not the numbers that count but the value of the project to the Services. It will not be out of place right now to tell you that I am not in favour of long term projects Research in the Armed Forces should have immediate operational value or be of sound clinical importance for those whom we serve. I concede a certain amount of fundamental research is essential but this should be restricted only to a category of workers who can devote the best part of their careers to it.

While I do not wish to interfere in your research projects, I must make it very clear that you must be careful in planning, so that sudden and repeated demands for subjects are not annoying for Service Headquarters ; wherever possible, several groups of workers should use the same subjects at any one time, in order to conserve our demands for Jawans. Further, we should not accept any projects which may lower the morale of our troops or create ideas where they do not exist, especially psychiatric and psychological projects. Such problems should be referred to and dealt with by the Directorate of Psychological Research. Earlier I had mentioned how our research effort had increased recently, but I find that it is concentrated mainly in AFMC ; Army Hospital, Delhi Cantt ; I A M Bangalore ; and NMRTC, Bombay. Research is not the prerogative of a limited number of units nor that of a small band of specialist officers. I want to see more of our hospitals in Lucknow, Jullundur, Chandigarh, Calcutta, Pune, Bangalore, Jorhat and other places to come into it in a big way. The Directors of Medical Services of the Army, Navy and Air Force may now consider it appropriate to either pose or request their Deputies in the Commands to formulate research problems for units under their medical administration. Similarly, the Chief and Senior Consultants must seriously consider giving projects to the peripheral hospitals rather than concentrating all research only in one or two centres e.g. Delhi Cantt.

It has been my observation that a great majority of research projects have not been planned properly, in that, competent statisticians have not been consulted, review of literature is incomplete, the aims and objectives of the project are not clearly defined, no previous survey of the availability of materials and equipment in the country has been carried out. This had led to innumerable difficulties and unnecessary delays in the execution. The individual Service Research Advisory Panels must resolve these issues before putting up projects to be examined by the Expert Groups. Further, the final reports I have seen leave much to be desired, and certainly many of them are not of a standard to be circulated. I hope the Directors of Medical Services will look into these problems.

In so far as the equipment is concerned, the tendency seems to be to want to obtain it from abroad. I am very averse to this idea, because not only are we short of foreign exchange but the instrument industry in the country will never progress if you do not use their products and advise them appropriately about their defects or improvements required. I have no objection to any unit or a specialist department building up its laboratory facilities with AFMRC equipment but I do not like the selfish tendency of refusing others the use of this equipment, especially when they themselves are not utilising it fully. Research equipment is for common use of all Departments and Sections of a unit, so long as they know how to use it and are careful in handling it.

I have instructed the D M R to take stock of all equipment purchased from AFMRC grants and to issue orders that the annual progress report must be accompanied by a properly authorised stock taking audit board reports—you may take this as an advance warning to bring your ledgers. vouchers, and other paper work up-to-date immediately on conclusion of this Conference.

Further, I want to impress on you that research equipment is very expensive and we cannot afford to have the luxury of duplicating it in our Service organisations. I would like some system of centralised recording instituted, so that it can be utilised more profitably by as many as possible.

Another very distressing attitude noted by me has been the increasing demand for staff to conduct projects. In some cases projects have not commenced or made much progress for want of SSAs, JSAs, Lab Assts, Lab Techs, Animal Assts or even sweepers. Gentlemen, I want you to note that in a majority of our Service institutions the responsibilities of an officer cover research training and clinical work. In some organisations there are already sufficient staff employed on AFMRC projects but they are not fully utilised and I would like you to share them for work. Perhaps you are not aware that the employment of staff for AFMRC projects has to receive the approval of the Scientific Adviser and we do not have sufficient justification to convince him in each case. You should also know that they cannot be employed for more than 2 years without going to the Government. On various occassions requests have been made for posting Service personnel of various trade groups and even though the AFMRC have accepted these proposals, yet the Service Directorates have not been able to meet this commitment for the simple reason that such personnel are available only for and can be posted against establishment vacancies. As such, I want the Directors of the three Medical Services to guide this committee when such demands do come up in our discussions. The matter has since been discussed in detail by D M R with Medical Directorates concerned and they have expressed the inability to provide this additional enrolled staff.

I am very happy indeed to see our Naval establishments making progress in research work; they have started late ue to unavoidable cirumstances, but I have no doubts in mind that with more encouragement from all members of this Committee, they will catch up with other two Services.

It is also good to see the Dental Corps and the Military Nursing Service taking an interest in research activities. I am sure that in subsequent meetings they will play an ever increasing role in the wider interest of the Armed Forces.

I am also trying to reorganise the DG AFMS Research Pool with the intention of employing them on specific projects on completion of which they should return to their service institutions to continue to work under their senior staff. The tendency to come on the Pool under one pretext or the other or making it an alternative to study leave is not in the interest of research and must stop. I would like to see more Air Force and Naval doctors on this Pool.

You are all aware that INMAS and DIPAS have now been placed under my direct functional and administrative control. I want you all to utilise the very considerable and important facility they have for your research or clinical work. INMAS already has a cell in the Army Hospital and we are investigating the possibilities of DIPAS having a similar cell in this hospital for biochemical studies.

A case to the Government to enhance the financial powers of the DG AFMS to sanction project upto 1 lac and equipment to the value of Rs. 25,000/- has been submitted. Those of you who have dealt with cases having a financial implication will appreciate the problem in getting Government approval. However, we hope to succeed. Then we can enhance the financial powers of Commandant, AFMC; OC, INHS ASVINI; and AOC, IAM.

It was most encouraging this year to receive 11 papers for the Colonel Amir Chand Award–they were all of a very high standard and the Awards Committee found it difficult to award the first prize. The award has been split and awarded to:-

(a) Lt Col M S. Dash
(b) Maj I.S. Chohan
(c) Lt Col (Mrs) K. Balakrishnan

Incidentally, we have filed an appeal in the court to change the name of this Award from Colonel to General Amir Chand Award but like many other things, even this has taken one year. Again, I hope that with the endeavour of my staff this will materialise soon.

The Director of Research in Basic Sciences is with us today, whose cooperation and help have no doubt been responsible for the conducive manner in which my staff have been able to work with the R&D. I am sure the present trend will be sustained in years to come and while accepting our thanks I will request him also to convey to the Scientific Adviser our gratitude for his help and sympathetic attitude towards us at all times.

We have a long agenda ahead of us so I will request the Committee to waste no time in starting the discussions. I am also of the opinion that projects which have been rejected by the Expert Groups should not be taken up by us here, since they have already considered them at length. Further, research workers and other specialists present here are required to observe the proceedings and only express their opinion when specifically requested by me or a Committee member.

Finally, I want to inform all project workers that the staff of the D.M.R. will be available in this Library between 14,00 and 16,00 hours each day, for them to check the recorded decisions of this Committee on their projects, so that if there is any ambiguity or controversy about them, we will resolve it at the subsequent meeting. No further correspondence will be entertained about these decisions later.

Thank you.

NUCLEAR MEDICINE : PRESENT AND FUTURE TRENDS

BY

Dr. A. K. Sundaram, MD*

Nuclear Medicine is concerned with the use of artificially produced radioisotopes for diagnostic, therapeutic, and investigative purpose.

These radioisotopes are usually radioactive counterparts of many stable elements participating in the physiological processes of the body. When used as tracers, they enable study of living processes in dynamic terms.

The main medical applications of radioisotopes today are largely concerned with the following functions:—

(a) Assessment of the functional activity of an organ-which is done by measuring the amount and rate of accumulation of a radioisotope in the organ, e. g. uptake of radioiodine by the thyroid gland.

(b) Scintigraphy, where the distribution of a radionuclide in an organ is charted for evaluating the functional integrity of each part of the organ.

(c) Estimating body spaces and exchangeable electrolytes e. g. total body, blood volume, exchangeable sodium etc.

(d) Measuring blood flow to various regions and organs of the body e.g. cardiac output.

(e) For measuring absorption of various labelled nutrients e.g. vitamin B-12 etc.

(f) For in vitro estimation of hormones and vitamins e.g. insulin, growth hormone.

(g) For studying the kinetics of various blood cells.

(h) For knowing the metabolic turnover of physiological substances e. g. protein turnover.

(i) For therapy e. g. use of radioiodine in thyrotoxicosis and thyroid cancer, where this radioisotope is administered in high doses to destroy pathological cells.

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* Director, Biomedical Group, Bhabha Atomic Research Centre, Bombay.

Though artificially produced radioisotopes have been in use in medicine for over two decades and Nuclear Medicine is well established in advanced countries, its development in India.began with the growth of the atomic energy programme in the country. For an effective nuclear medicine programme in any country both radioisotopes and electronic instruments should be easily available. Self-sufficiency in this area has been achieved to a great extent in India, most of the clinically used radioisotopes being produced in the reactors at the Bhabha Atomic Research Centre (BARC), Trombay.

Production and distribution of radioisotopes on a commercial scale in the country was taken up by the Isotope Division of BARC in 1961. In the past 10 years, about 35 radiopharmaceuticals ; both in elemental and labelled form, have been made available by BARC to medical laboratories in India and abroad. India today has advanced capabilities in regard to the variety and quantity of radiopharmaceuticals that can be manufactured for medical applications.

The production of electronics instruments for medical purpose is done at the Electronics Corporation of India Ltd. a public sector undertaking of the Department of Atomic Energy, at Hyderabad. R & D effort at the Electronic Division of BARC, which is a poineer in this line, is also contributing.

At present, the country has about 73 radioisostope laboratories, most of them attached to general hospitals. There are two large Nuclear Medicine Institutions: the Radiation Medicine Centre of the Bhabha Atomic Research Centre, and the Institute of Nuclear Medicine and Allied Sciences of the Ministry of Defence at New Delhi, besides these two, many outstanding medical institutions such as the All India Institute of Medical Science, New Delhi, the Christian Medical College & Hospital, Vellore, the Banaras Hindu University and most medical colleges of Bombay have radioisotope laboratories.

In 1963, soon after the supply of radiopharmaceuticals on a routine basis became possible, the Department of Atomic Energy established the Radiation Medicine Centre (RMC) at Bombay. In the last 10 years the RMC has come to enjoy both national and international recognition. Recently, both the World Health Organisation and the International Atomic Energy Agency have recognised it as a Regional Reference Centre for South East Asia in the field of Nuclear Medicine.

The RMC provides free service facilities for investigations and treatment with radioisotopes primarily to create awareness in the medical fraternity of the potentialities of Nuclear Medicine. Nearly 10,000 patients avail of this Service annually.

The RMC also offers training programme for building up medical and paramedical personnel. For the last 8 years, it has conducted a 6 weeks basic training course on "Medical Uses of Radioisotopes" every year. So for a total of 113 trainees have taken advantage of these courses. The Centre has also a short advanced and intensive course on a specific clinical radioisotope technique every year. The University of Bombay has accorded recognition to the 'Diploma in Radiation Medicine' for medical graduates and 'Diploma in Medical Radioisotope Technique' for science graduates, conducted by Bhabha Atomic Research Centre. The University of Delhi is another university which has recognised this discipline. A one year academic programme leading to these qualifications has been started at the Radiation Medicine Centre this year.

The number of radioisotopic investigation per year per 1000 of the population in Greater Bombay works out to 2 per 1000, a figure comparable to that in Japan and New Zealand. This favourable figure for Bombay has become possible because of the facilities at the Centre and also because of the training programme available to the doctors in this region. Besides the Radiation Medicine Centre, Bombay has seven other Nuclear Medicine Units. Of course, all the four teaching, medical institutions in the city have their own radioisotope laboratories. Some more, well equipped radioisotope laboratories are expected to come up in the near future.

In the last few years, there has been a major shift in the trend of medical applications of radioisotopes. This has been mostly in scintigraphy. The trend now is to use short half-life generator-produced radionuclides. Precise quantitative information about the functions of many endocrine glands is obtained by in vitro procedures like radioimmunoassay. The increasing use of this has resulted in considerable reduction of radiation exposure of the patient during diagnosis.

With the use of the short half-life radioisotopes, the radiation exposure of the patient is negligible which makes possible extension of radioisotopic investigations to children and pregnant women, the two groups which were hitherto not covered because of radiation risks involved.

Along with the use of short half-life radioisotopes, the character and design of the scintigraphic equipment has changed. The modern scanner accomplishes a scan image of a large organ at a speed which is 5 to 10 times higher than what was possible with the scanners in the sixties. This speed is possible because an optimum amount of radioactivity can be administered to a patient without undue risk as the radioisotopes used have a short-half-life. This is of special significance to our country as faster scanning techniques permit effective handling of a larger number of patients in a shorter time.

CYCLOTRON-PRODUCED RADIOISOTOPES

The use of cyclotron—produced radioisotopes has just started in a routine way at many centre, in the world. Radioisotopes which show promise in this respect are : ^{18}F, ^{123}X, ^{67}Ga, ^{15}O, ^{13}N, ^{111}In etc. ^{131}I, the most widely used radioisotope at present has several undesirable physical characteristics. With the increasing availability of ^{123}I, the use of ^{131}I, for diagnostic applications will gradually decrease. When the variable Energy Cyclotron at Calcutta became operational, it may become possible to undertake production of some radioisotopes which have a relatively longer half-life.

^{133}Xe can be produced both in the reactor and in the cyclotron, but it is in gaseous form at the time of irratiation. It can be subsequently dissolved under pressure in normal saline solution for clinical use. Its use is gradually gaining recognition for lung function studies. There could be wide-spread demand for it in India because of the multitude of respiratory problems in the country.

The next decade may well be said to belong to radioimmunoassays. These and similar procedures based on the principle of saturation analysis are done in vitro, without administering any radioactivity, and with a minimum of instrumentation, to obtain precise quantitative measurement of blood levels, hormones, vitamins and other important physiological parameters. ^{125}I is used instead of ^{131}I because it gives long-life of the labelled compound and because its use involves less radiation hazard. Radioimmunoassay technique however requires scrupulous care and high technical skill.

FUTURE NEEDS

The greatest contribution of Nuclear Medicine is to diagnosis. Diagnostic uses of radioisotopes cover a wide field and embrace almost all the clinical disciplines. Some of the diagnostic procedures are simple and some extremely complex. The range at present covered in our country is restricted to simple procedures and only a few laboratories here have achieved any degree of sophistication in their work. There are many clinical problems specific to this country where radioisotopes would provide great help. However, very few laboratories have attempted to look beyond the conventional thyriod problems.

It is obvious that for future expansion of nuclear medicine in this country, existing radioisotopes laboratories will have to enlarge the range of their work and many more laboratories will have to come up to exploit the benefits offered by radioisotopes in medical diagnosis and research.

It will be economical and efficient if small radioisotope laboratories attached to teaching hospitals function as centralised units with persons working full time in the area of Nuclear Medicine.

In addition to these small hospitals radioisotope laboratories, the country would need more centres like the Radiation Medicine Centre of BARC to undertake the full range of diagnostic and therapeutic applications of radioisotopes, to provide training to medical and paramedical personnel and to develop new procedures and research into clinical and associated fields of nuclear medicine.

Exposure to radiations has a definite risk both to the patient, the doctor and the general public. While hazards from conventional techniques are easily identifiable, those arising from improper and indiscriminate use of radiations and radioisotopes may come up after a number of years and same, in future generations. It is therefore essential that the medical doctors and paramedical staff are stringently certified after an extensive theoretical and practical training in the various aspects of the application of radioisotopes to human being. The institution of a diploma in nuclear medicine for medical doctors and a similar diploma for non-medical personnel by the University of Bombay is an outcome of the special requirements of this relatively new technology.

If we aim at having one nuclear medicine department in every hospital in the country, the demand for trained personnel is going to be high, and training programme should be increased and made available in a large number of centres.

THE CLINICAL APPROACH TO "MODERN CONCEPT OF EARLY DIAGNOSIS AND ITS VALUE IN THE MANAGEMENT OF DIABETES MELLITUS"

BY

Dr. SAM G. P. Moses*

Ladies and Gentlemen,

I feel proud and honoured to have been requested to deliver a Guest lecture in this subject to this large enlightened audience. I propose to put across a few ideas in this connection mainly under four headings:-

(a) Few concepts and applied aspects in Clinical Diabetology in the present day understanding.

(b) Early diagnosis of Diabetes Mellitus mainly derived from:

  (i) Clinical stigmata

  (ii) Various metabolic tests of real practical value and

  (iii) A few tests for the vascular assessment.

(c) A small note and recognition of abnormal GTT and blood sugar levels in otherwise healthy young adults with few basic considerations in relation to nonalertness and risk factors in them.

(d) A few random aspect about the management and as to what to do with the early detected Diabetics.

FEW CONCEPTS AND APPLIED ASPECT IN CLINICAL DIABETOLOGY IN THE PRESENT DAY UNDERSTANDING

Diabetes mellitus is a metabolic cum vascular disease due to an inborn error of carbohydrate, Protein and Fat metabolism due to absolute or relative insufficiency of insulin action with a special tendency for vascular disease. The speciality of Diabetology is well known with the clinical, experimental and biochemical aspects as the three important divisions. It is also becoming increasingly recognised that Diabetes is an important single disease speciality of special significance to the physician and to the patient since he has to be educated to well live with it.

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In Clinical Medicine, the diabetics met with by physicians in day to day work could be classified as :-

(a) Maturity onset diabetics with enough endogenous insulin as most frequently seen.

(b) Younger age group diabetics with very little or no endogenous insulin and often insulin dependent and.

(c) Rare group of secondary Diabetic syndromes where diabetes occurs in addition to other group of problems Cushing's acromegaly etc.

The younger age group diabetics have been of particular interest to us well as to many other diabetologists of India since marked differences in the pattern do occur in this age groups (30 years and below) in different parts of the country as when compared with the Western World but in maturity onset diabetes, this may not be the case. Thus over a long term study and observations six different patterns of these yonger age group diabetics have been recognised :-

(a) Classical brittle type (20%)

(b) Nonketotic, thin, insulin indepndent diabetics mainly from the poorer strata (50%)

(c) A small merger or intermediate group.

(d) A small minor percentage of maturity onset type in younger age group with slight obesity and occasional response to oral drug.

(e) A small number of rarer juvenile diabetic syndromes like mauriac, Alstrom's Werner, Refsum's syndrome etc. of genetic origin and.

(f) A separate significant group of less than 25% of these cases grouped as pancreatic type with pancreatic calculi and calcification often endemic in areas of Madras and Kerala and rarely seen in the North.

A few slides illustrating these patterns from our Department of Metabolism and Diabetology, are shown here.

It is but natural in this disease that a series of genetic syndromes associated with carbohydrate intolerance have been enlisted by Rimoin and even grouped in an alphabetical order reminding the physician about these rare syndromes.

Next I may probably reflect upon the essence of what the Clincian should know of blood sugar methods. The 'true glucose and total" reducing substances, whole blood Vs plasma glucose level; venous and capillary levels of glucose as also the methods, of presrvation, time of withdrawal of blood and the performance of the tests.

The pros and cons of the various blood sugar methods have been emphasised by many authors and today the glucose oxidase, Somogyi Nelson, Hoffman's auto analyser and lastly the ortho tolindine method (popularised by Eastern authors) and the various modifications are all familiar to physicians.

The Clinician's look at the G.T.T. and its applications are well known. The fasting level, the earlier half of GTT denoting the absorption and the latter half (after the peak) denoting the disposal of glucose and the normal standards are all to be well, known by the physician. Although Fajan's Criteria are well accepted, individual authors are bound to have slight difference with valid reasons of their own. Although we have formulated our own criteria, it is advisable for each group of diabetologists to set their criteria on their own population.

The various modifications of the GTT like 50, 75 or 100 GM challenge dose, Extended GTTS (3 to 5 hours) in special instances, and the diurnol variation of the GTT (so called morning and afternoon GTT) to diagnose early abnormalities, and lastly the cortisone primed GTT are to be familiarised especially in the concept of early diagnosis. The intravenous GTT however has not been found to be of practical value, though sometimes employed. On the other hand, the other parameters like free fatty acid and Insulin levels during GTT are of great significance and are helpful diagnostic tools. The exact method preparation for a GTT with 3000 calorie diet and the standard specifications are very well known.

An important set of principal nondiabetic causes of abnormal GTT and hyperglycemia like liver disease, chronic illnesses, bed rest, endocrinopathies, Uremia etc.are all very well known. Other factors influencing oral GTT like age, sex, menstrual cycle, emotions or even posture during the tests are also to be remembered. Other associated diseases and conditions which may modify the GTT like pregnancy (gestational diabetes) gout and other metabolic disorders, stress, trauma etc. have also to be remembered in the interpretations. In addition surgical hyperglycemic temporary states like burns, anesthesia hypoxia, Cardiopulmonary by pass are also to be occasionally considered. The concept and occurence of intermittent carbohydrate intolerance during observation for long periods over many years occuring even in younger diabetics, has been very impressive in this author's practical experience also thus adding to the problem of assessing or calling it a "hyperglycemic" or "Diabetic" state.

Iatrogenic or drug induced alterations in the blood sugar level and GTT are well known. Most drugs worsen carbohydrate metabolism like cortisone, oral contraceptives etc while salicylates Atromid S, alcohol and sulphonamides may lower blood sugar levels. In clinical practice, alcohol is a powerful hypoglycemic agent especially in an empty stomach and in the author's impression is of great significance.

Ageing and deterioration of carbohydrate tolerance has been well documented leading us to wonder whether we should alter the criteria of normality of blood sugar levels over the age of 50m years and already nomograms are available. In clinical experience, one gets an impression that there are two sets of older individuals one truely hyperglycemics for long periods without diabetic problem and secondly true diabetics with diabetic problems-mixed up in the general population. This may be superficially compared to osteoporosis in the physiological form reaching pathological proportions in the same age groups.

The Beta cell has assumed very great clinical significance since last three years and the Biochemical concepts of the pathogenesis of Diabetes revolves on this more than peripheral mechanisms such as insulin antagonists or resistance to circulating insulin etc. so much so a very large work has been done by Lacy and his colleagues. The granulation in the Beta Cell and release of Insulin and the small halo of excorine pancreatic tissue around the isolet being of equal importance as much as the Beta Cell have all to be considered as important advances especially in the present context of Beta Cell perfusion and transplantations.

Naturally, the insulin releasing factors have assumed great importance in pathogenesis and management. Apart from glucose, aminoacids, electrolytes (Ca.ion and Mg.ion) and drugs may all play a great role in day to day insulin release and offers food for thought for at least next few years in this field.

The insulin antagonism and the resistance has been very well known since the Vallence Owens ideas. But this may not play a large part in all the cases. The Central B cell blockade, circulating insulin antibodies and lastly the peripheral insulin antagonism are all diverse subjects of great biochemical importance.

The vascular component namely specific microangiopathy and its biochemical study has made great progress as also the thickening of basement membrane most frequently seen in diabetics. But what is more important is recognition of these changes in pre-diabetics. Macrovascular disease and exaggerated atheroma are all well known.

The connecting link between the metabolic and vascular component has been the hue and cry in Diabetology and a large amount of biochemical research is being done in this direction. Mucopolysaccharide, Blipoproteins, and the Sorbitol pathway definitely playing a great role in metabolic cataract as well as neuropathies, insulin antibodies to exogenous insulin have all been frequently blamed as the culprits and Diabetologists from various quarters are anxious to build up the connecting links so as to be of positive help to offer direct treatment for microangiopathies which is now lacking except occasional claims.

EARLY DIAGNOSIS OF DIABETES MELLITUS

Now reflecting on the second aspect of the lecture-namely the early diagnosis of diabetes mellitus, it is possible today to diagnose an early diabetic state from three different aspects namely clinical stigimata, metabolic component tests and test for the vascular components. All these three aspects could be used for assesment and follow up to be of benefit in the management.

The various clinical stigmata and back ground phenomena such as asymptomatic parotid glandular enlargement (although very highly nonspecific), Dupytren's Contracture, segmental calcification of vessels, segmental neuropathies and (Visceral), progressive oversized babies in pregnancy, gross and vascular changes in placenta, Xanthomas, dermopathies, premature atherosclerosis, recurrent balanoposthitis etc. may all be fore runners. The occurence of this abnormality in other members of family may also be significant. Spontaneous or reactive hypoglycemia is the most well recognised and convincing clinical evidence of early diabetic state especially if it is provable by extended GTT.

A variety of metabolic tests are now definitely available for early detection. Routine screening with 75 Gm or 100 Gm glucose load, single two hours blood sugar level, and the GTT, may quickly help for the detection. In addition, the extended GTT no doubt is most valuable in reactive hypoglycemia. The so called morning and afternoon GTT if done with good normal controls would be of additional value. The Insulin assay run parallel with GTT will reveal the initial blunting which is so characteristic of diabetes whereas the two peak response is characteristic of normality. The lipoprotein pattern, and Fredrickson's Types III and IV may suggest the oncoming carbohydrate intolerance. Although all these tests are available, a repitition of them at regular intervals may be much more helpful.

Cortisone primed GTT is somehow not often used routinely for the detection of early chemical diabetic state. It is however most useful for confirming the pre-diabetic-state whenever the Clinical situation warrants as for example fetal wastage in pregnancies, diabetic phenomena rarely occuring the prediabetic state (retinopathy, nephropathy etc). In Clinical practice, prediabetic state is very often a retrospective diagnosis. Further practising clinicians do not often require the necessity of diagnosing prediabetic states. Again, rarely few diabetologists have reported the precipitation of the evident chemical diabetic state on trying out corticosteriods, although is difficult to be believed. So while there may be no direct objection to use cortisons GTT routinely, it may not be very wise as a practical step and hence, has not been recommended universally. Further in the newer way of thinking, we will be unnecessarily dubbing a number of individuals prediabetes without having much to offer them.

Since diabetes is a metabolic cum vascular disease. Early diagnosis should assess the vascular component also without which it will be incomplete. The basement membrane thickening of thigh muscles, skin and gums and occasionally internal organ biopsies (Conjunctiva, Kidney etc.) or by reviewing the placenta during pregnancies may all be recommended. In addition of course, routine clinical vascular survey to detect any abnormality should be carried out like segmental arteriopathy may be of corroborative value. An added novelty to this armamentorium is the concept of functional microangiopathy by determining blood flow in small area apart from histopathological changes in live tissues may add to the list.

A SMALL NOTE AND RECOGNITION OF ABNORMAL GTT AND BLOOD SUGAR LEVELS IN OTHERWISE HEALTHY YOUNG ADULTS WITH FEW BASIC CONSIDERATIONS IN RELATION TO NONALERTNESS AND RISK FACTORS IN THEM.

Now reflecting on the third part of lecture, abnormal blood sugar levels and GTT in otherwise healthy young adults, probably a few basic considerations and non alertness factors and risk factors may be taken into consideration. It is common agreement that diabetes is inherited and it is also well known that vascular component may precede by many years the metabolic abnormality. It is another fact that recognisable carbohydrate abnormality in GTT may precede clinical diabetes by even decades. Further abnormal GTT and even asymptomatic chemical diabetes need not show progression of carbohydrate intolerance and live a normal life span and die without any diabetic problem. It is a well known fact that even an intermittent nature of glucose intolerance is well recognised and has been even observed by us. Finally, even in otherwise normal individuals, it is well known that there can be variations in the glucose tolerance in the same individual if repeated at intervals under same conditions.

The risk factors and 'Non-alertness' in healthy young adults with abnormal GTT is probably of great concern to physicians in certain environments like aviation medicine and other selected communities. In this connection in the author's opinion spontaneous reactive hypoglycemia which is well known early symptom whether diet induced, amino-acid induced or alcohol induced may be of practical significance but apart from this, premature vascular problems need not themselves pose a great handicap. Again psychiatric upsets about interpretation and such diagnosis and irritability of the candidates leading on to lack of confidence in the medical personnel and other social phenomena have to be taken note of. Finally an exaggerated over diagnosis may lead to attribution of several different symptoms to this event.

A FEW RANDOM ASPECTS ABOUT THE MANAGEMENT OR AS TO WHAT TO DO WITH THE EARLY DETECTED DIABETICS

Lastly if one decides to ponder about the management and as to what to do with the person with abnormal GTT after exclusion of oher causes clinically and by investigation, one really gets baffled because of our very little armamentorium. What to do with them may also depend on their employment, environment, medical personnel etc. Probably it will be advisable to repeat the GTT at interval for three times and regular follow up once in three months for 3-5 years with vascular survey and follow up of microangiopathic studies and lipoprotein abnormalities. Another aspect would be to check up the close relatives of these subjects for simliar abnormalities. It is also advisable to do several comparative group studies of such nature with similar groups elsewhere also. It is also advisable to have group discussions with specialities about this problem so that one way decision need not be taken.

It is generally agreed that institution of proper diabetic regime is warranted on detection and to make the individual convinced of the changes in diet, exercise and other hygienic measures and the avoidance of repeated pregnancies, avoidance of unnecessary drugs (steroids oral contraceptives etc.), may all have its value. Only very few diabetologists believe in the display of oral antidiabetic agents like phenformin (DBI) which may be useful for short periods. Other sulphonylureas are rarely used. While medical advice to include dietitic regime protein extracts and correction of Calcium deficiency may have theoretical considerations. It is no doubt that obesity has to be controlled or prevented and anorectic drugs like fen fluramine may have insulin sparing value.

Before we conclude, I have to say that there are very many recent advances in Clinical Diabetology, biochemical diabetology and experimental diabetology and what has most impressed me is the discovery of the electronic implantable glucose sensor mechanism enabling the continuous glucose monitoring of glucose levels. Even more it is thrilling to find the discovery of artificial beta cell with an implantable Insulin Reservoir beneath the skin to enable Insulin refuelling over a long period of time. One wonders whether this will not be a useful physiological substitute for the daily injections.

Now that this discovery has set the ball rolling, many such discoveries will appear in this field.

Finally all good things have to come to an end so I have to conclude that I have had the great pleasure of being able to put across a few ideas as they crossed my mind in this field of my interest over a number of years. I have only to say that we look forward to many new advances in this field in our lifetime in the years to come.

Thank you

P 05.162

INTRODUCTION BY THE CHAIRMAN

BY

Maj Gen R.S. Hoon, Sen. Con (M)

The seminars and symposia held during the past few years have exhaustively dealt with some of the medical problems confronting the Armed Forces Medical Services. Ischaemic Heart Disease, Peptic Ulcer, Diabetes Mellitus, Pleural Effusion and Effects of High Altitude and Cold are important examples. Of the remaining diseases Malaria, Viral Hepatitis, Amoebiasis and Hazards of Drug Therapy hold the promise of wide interest and application. The four conditions collectively account for 1/20th of the total mortality amongst the services. In respect of individual diseases, the following features merit detailed discussion:

(a)	**Malaria**	The breakthrough in the National Malaria Eradication Programme with resulting reappearance of cases and of resistance to antimalarial drugs/insecticides.
(b)	**Viral Hepatitis**	The rising incidence, the study of Australia Antigen amongst patients, carriers and contacts and correlative analysis of clinical, biochemical and histological data.
(c)	**Amoebiasis**	The protein manifestations of hepatic amoebiasis and the methods for early diagnosis and safe/effective management.
(d)	**Hazards of Drug Therapy**	The plethora of pharmaceutical preparations, the explosion of literature seeking to promote their uninhibited administration and an appraisal of the scope and limitation of these agents in modern medical practice.

Selected specialists will present their knowledge and experience in the respective fields in the following orders:

Malaria

Preventive Aspect	Brig M.C. Sanyal Prof of Prev. Med; AFMC

Diagnosis and Management of cases and complications	Col A.R. Subramanian Sen. Adviser in Medicine, Eastern Command

Viral Hepatitis

Laboratory Aspect	Col R.N. Dutta, OC AFTC
Clinical Aspect	LT Col B.B. Nanda, Specialists in Medicine, Base Hospital
Epidemiology	LT Col R.N. Varma, ADG (H), Office of DGAFMS

Amoebiasis

Medical Aspect	Surg Capt C Kuppuswamy, Sen. Adv. in Med., AF Hospital, Bangalore
Surgical Aspect	LT Col G.S. Srivastava, Specialist in Surgery, Army Hospital

Hazards of Drug Therapy

General Introduction	Brig S.N. Basu Prof of Med., AFMC
The Toxicity & Sensitivity Reactions	Col K.C. Gupta, Sen. Adv. in Medicine, Northern Command
The Drug Resistance	Col D.S. Rastogi, Sen. Adv. in Dis. of Chest MH (C. TH. C) Poona
The Role of a Nurse in Recognition and management of Drugs Reactions	Lt (Miss) P.N. Rajeswaryamma, MNS Command Hospital, Poona

CURRENT MEDICAL PROBLEMS : PREVENTIVE ASPECTS OF MALARIA IN ARMED FORCES

BY

Brig M.C. Sanyal*

INTRODUCTION

Malaria prevention in the Armed Forces should be considered in relation to the total picture of malaria in India. This picture is now complicated by the factors of recrudescence, vector resistance to insecticide, and early parasite resistance to antimalarial drugs. An additional recent factor is the likely difficulty in procurement and supply of anti-larval oils. The resultant problem in the prevention of malaria in a service population is further varied by whether that population is serving in a 'peace' station or in a 'field area'.

EPIDEMIOLOGY

Malaria in the Army

At the beginning of this century malaria morbidity in the Indian Army was 320.8/1000 in 1900 and in 1949 it was 30.4/1000. By 1938 antilarval measures had brought the incidence down to 86.7/1000 in peace stations but operations in the frontier did cause peaks in the rate. World War II saw a rise in the malaria rate when the largest number of admissions into hospitals was from this disease. In SEAC malaria was the cause of nearly half of the total sickness during 1942-44 and at times during the main transmission season it accounted for more than 80% of all admissions. In the Arakan campaign of 1943 some of the units were so depleted of their personnel by malaria that replacements had to be provided every 6-8 weeks.

A decline in the number of cases commenced in 1943 when mepacrine suppression was started after Hamilton Fairley demonstrated the military value of the drug. It was conclusively proved that mepacrine taken regularly suppresses malaria. The "mepacrine parade" under the supervision of an officer, the detection of mepacrine in the urine, and the laying of responsibility for "break through" on unit commanders had a salutary effect. But it was also realised that as the necessity for suppression indicated a high risk

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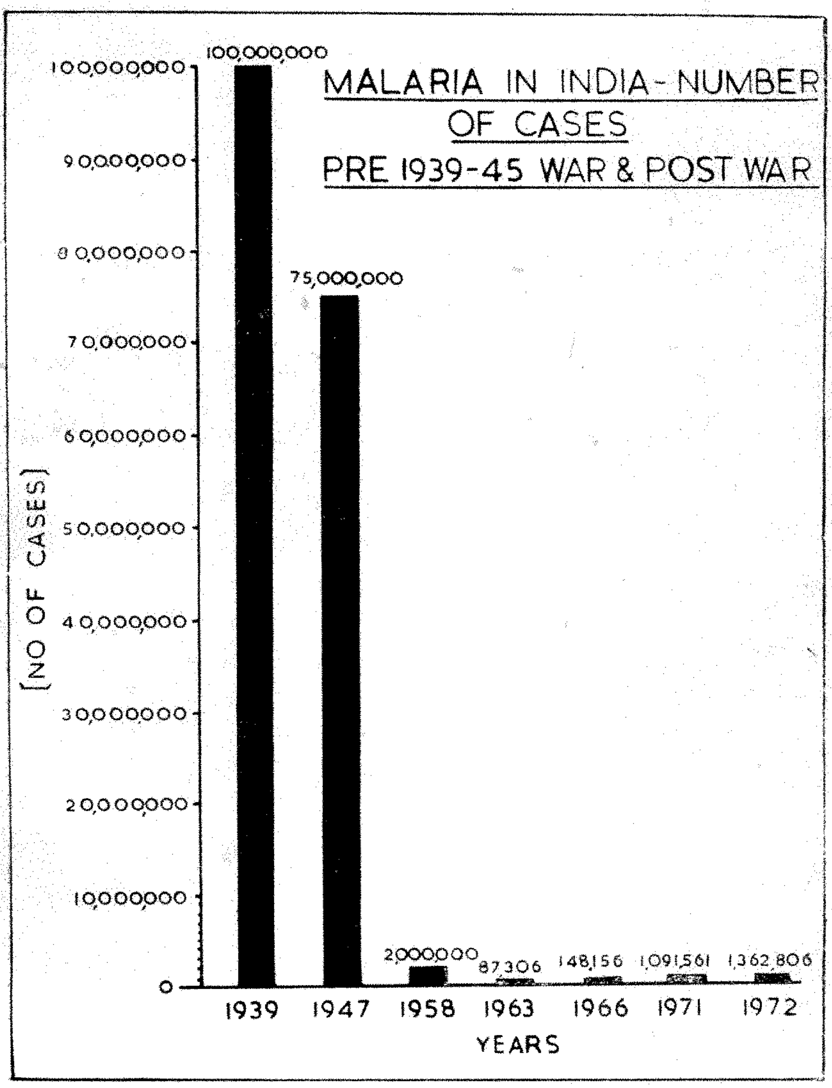

Fig. 1—Malaria morbidity in the Indian Army 1900 to 1972.

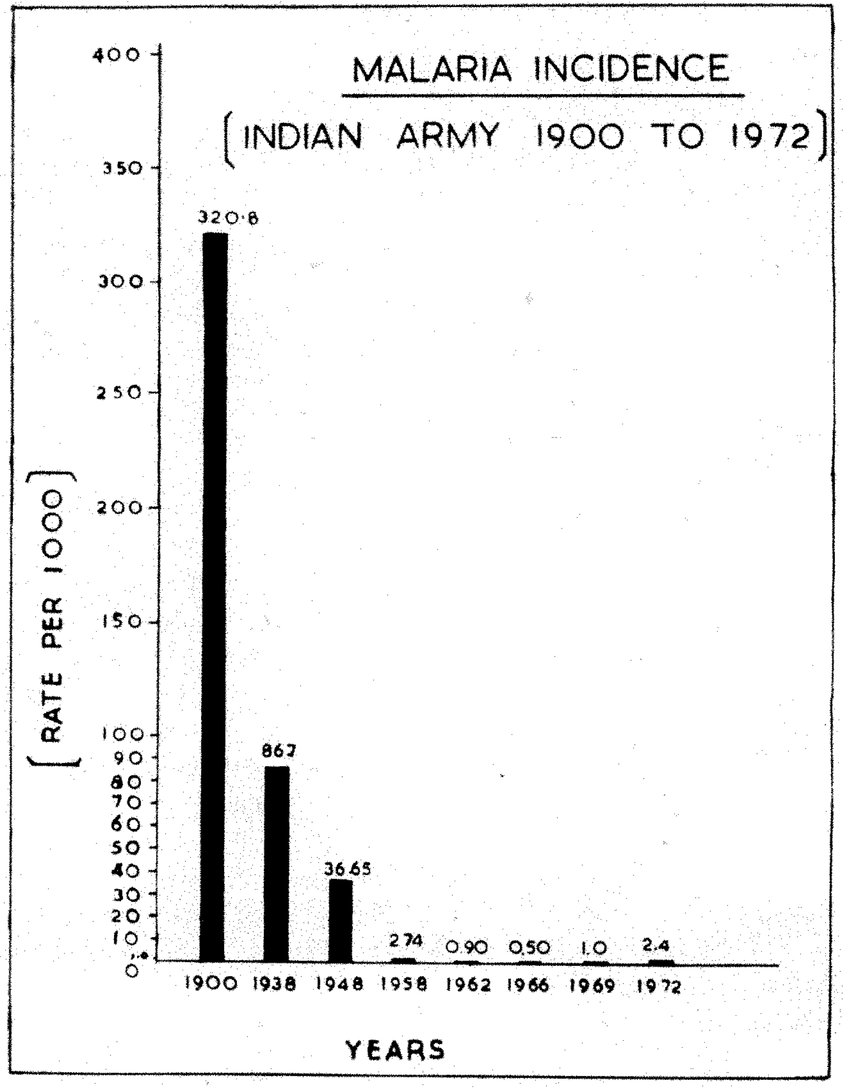

Fig. 2—Incidence of Malaria in Civil Population.

of malaria, efficient personal and unit protective measures were also of paramount importance (Raina & Prasad, 1955).

In 1948 the rate was 36.65/1000 and by 1958 it was 3.24. Thereafter it fell to 1.60 in 1961 and from then onwards the rate fluctuated between 0.50 and 2.40.

Malaria in Civil Population

Before World War II the national estimate for malaria in India in ordinary years was thought to be around 100 million every year with an annual mortality of 1,000,000. In 1947 the estimate was 75 millions suffering from the disease with an annual mortality of about 800,000. In 1958 (Govt. of India 1958) when the NMEP started their campaign 2000,000 cases were estimated. In 1963, 5 years after the start of the malaria eradication programme, the number of cases was 87,306. In 1966 there was a setback and 148,156 cases were recorded. Thereafter the number of cases increased annually and in 1972–1,362,806 cases were recorded.

The epidemiology that we are now seeing is that of disappearing malaria during an eradication programme. That is recrudescent malaria from failure of surveillance and persistent malaria either from failure of eradication measures, vector resistance to insecticides or parasite resistance to anti-malarial drugs or a combination of all three factors. The question now before us is whether the objective of eradication is possible or not. This matter is currently being considered by a committee.

The problems in the prevention of malaria in the Armed Forces should be viewed against this background.

PREVENTION OF MALARIA IN "PEACE" STATIONS

In "peace" stations located in areas where malaria has reappeared or where it has not been eradicated the problems in the Armed Forces are of enforcing personal preventive measures, of providing a good insecticide cover, conducting an efficient anti-larval campaign and detecting residual cases of malaria.

Enforcing personal protective measures is the responsibility of commanders and where they are not enforced it is a failure of command. The responsibility of the Services in HQs is to ensure that the stores required are provided in right quantities at the right time. Such stores include mosquito nets, spray equipment, insecticides, anti-larval agents and insect repellents.

The medical responsibility is to prod staff to enforce protective measures and to position stores. It is also their responsibility to carry out systematic anti-larval operations and to train non-medical and para-medical personnel in the use of the various measures. It is also their function to diagnose malaria and treat it radically when diagnosed.

In "peace" stations suppression has a limited place in the prevention of malaria. If it has to be used it must be as an adjunct to good insecticide cover and efficient anti-larval operations. The situation when suppression may be ordered in a peace station is in an area where malaria transmission is prolonged and is mainly falciparum. In such a situation there must not be any exceptions to the mass administration of the drug and that is the problem. To administer the drug to all persons regularly requires a degree of organization and discipline which is possible only in a service context and even under these conditions there are failures.

PREVENTION OF MALARIA IN "FIELD AREAS".

In areas where malaria transmission has not been interrupted, apart from the rigid enforcement of personal preventive measures and insecticides cover in living accommodation, malaria suppression has to be instituted. The problems posed arise from the operational situation. In the circumstances where troops have to be located in detachments in hostile country and are required to guard their perimeter at night and patrol in their zone of control there is likely to be irregular suppression for a variety of reasons—inadequate supervision, unexpected prolongation of patrols, intermittent supply of drugs. Under these conditions, in an area of intense transmission, when weekly suppressive doses of antimalarial drugs are sometimes missed there will be bursts of parasitaemia in individuals causing mild fever which go unrecognised as malaria. In the absence of laboratory facilities such attacks are generally treated with a combination of antipyretic drugs, and chloroquine in suppressive doses with which the fever subsides leaving some of the parasites in the peripheral blood unharmed. There are successive episodes of mild fever, inadequately treated with suppressive doses of chloroquine, leaving more and more malaria parasites unharmed until their level in the peripheral blood rises to a pitch when a full blown attack of malaria manifests. The soldier is then evacuated to hospital where very often he does not respond adequately to the standard treatment of 600 mgm of chloroquine and 75 mgm of primaquine exhibited over 5 days and the physician is left wondering whether he is dealing with a 'resistant' malaria or a recrudescent malaria.

NEW PROBLEMS.

The two new problems which are of rising importance are:

(a) Vector resistance to insecticides.

(b) Parasite resistance to anti-malarials.

(a) Vector Resistance

Of the important anopheline vectors of malaria in India insecticide resistance to DDT has been demonstrated in *A. culicifacies* and *A. stephensi* in a few areas. For the time being however the organophosphorus group of insecticides are effective. But we will soon be out of replacements. Therefore anti-larvel measures to control and eliminate mosquito breeding in urban areas needs greater emphasis especially permanent engineering control and elimination of mosquito breeding. Biological control also offers new possibilities. Mosquito control to-day is in a state of crisis because of our excessive reliance in the past on organic insecticides.

(b) Parasite Resistance

In all areas of South East Asia where parasite resistance to antimalarial drugs has been marked there have been common factors in the prevailing circumstances. Unstable endemic malaria situations have been usual; migration of populations have been common and chloroquine has been readily available (WHO, 1967).

The primary factors in the causation are under-dosage and significant parasitaemia, operating together found particularly in the under-treated acute attack and in the under-suppressed smouldering infection. In suppressive therapy with missed doses parasitaemia may rise to a clinical level and the dual effect of a high concentration of parasites and a low concentration of drug come into paly (Covell, Coatney, Field and Singh, 1955).

In India falciparum resistance has been confirmed in two places in the Mikir Hills and is suspected in Mizoram. In these areas the factors mentioned earlier conducive to the development of parasite resistance are present. It is likely that more areas may be discovered where there is early resistance. It is a possibility of which we must be constantly aware, but not to be panicked about.

PROCEDURES FOR ASSESSING DRUG RESISTANCE

The procedures for assessing drug resistance are given in WHO Tech. Rep. Ser. No. 529 (WHO 1973) reproduced below.

"The first step in assessing drug response is to collect baseline data on the sensitivity of *P. falciparum* to chloroquine not only in localities from which reports of suspected resistance have been received but also from areas of distribution of this parasite where the drug response appears to be normal. Several tests are available. The options available are the following :-

(1) The WHO Standard Field Test consisting of the administration of 25 mg of chloroquine base per kilogram of body weight over 3 days, with 7-day observation period (sometimes referred to as the "7-day test").

(2) The same test with the observation period extended to a total of 28 days, referred to as the "extended test".

(3) The single-dose test, or "alternative test" , consisting of the administration of 10 mg of chloroquine base per kilogram of body weight. This test is applicable:

(a) where for any reason treatment cannot be pursued for 3 days;

(b) in areas of high endemicity where, owing to the elevated level of immunity in the population, a single dose of chloroquine has been accepted as the standard form of treatment; or

(c) as a preliminary screening procedure prior to applying the standard 3-day treatment."

INTERPRETATION OF THE W H O STANDARD FIELD TEST (7-DAY TEST)

"This test is interpreted as foollows :-

(a) If no asexual parasites are found by day 6 and none are present on day 7, the infection may be either sensitive (S) or resistant at the RI level.

(b) If asexual parasites disappear for at least 2 consecutive days but return and are present on day 7, they are resistant at the RI level.

(c) If asexual parasitaemia does not clear but is reduced to 25% or less of the original pre-test level during the first 48 hours of treatment, the parasites are resistant at the RII level.

(d) If asexual parasitaemia is reduced by less than 75% during the first 48 hours or if it continues to rise, the parasites are resistant to the standard dose of the drug at the RIII level."

INTERPRETATION OF AN EXTENDED TEST

"This test will distinguish between sensitivity (S) and the kind of resistance that is demonstrated only by recrudescence following a normal initial response. It is interpreted as follows :

(a) If no asexual parasites are found by day 6 and parasites do not reappear by day 28, the parasites are sensitive (S).

(b) If asexual parasites disappear as in (a) but return within 28 days, reinfection having been excluded, the parasites are resistant at the RI level.

(c) If the asexual parasitaemia does not clear but is reduced to 25% or less of the original pre-test level during the first 48 hours of treatment, the parasites are resistant at the RII level.

(d) If asexual parasitaemia is reduced by less than 75% during the first 48 hours or if it continues to rise, the parasites are resistant at the RIII level."

ALTERNATIVE TEST WITH SINGLE-DOSE TREATMENT

"An alternative test using a single dose of 10 mg of chloroquine base per kilogram of body weight can be utilised instead of the 3-day regimen. The observation period is limited to 7 days and the test is interpreted as in the WHO Standard Field Test. However, should the parasitaemia fail to respond within 7 days of recrudescence during this time, the 3-day regimen should be applied."

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DIAGNOSIS, COMPLICATIONS AND MANAGEMENT OF MALARIA

BY

Col A.R. Subramanian*

INTRODUCTION

1 In the early part of this century Malaria used to be one of the common causes of fever in our country and its incidence was quite high in the Armed Forces. However, intensive entomological control of the vectors, personal protection measures and chemo-suppression had helped to bring down the incidence considerably over the years and one saw very few cases of malaria in the fifties and sixties. The situation has changed once again in the recent years. Renewed interest and concern has been created especially in the Eastern Sector where two fresh problems have cropped up. Firstly, there has been a definite rise in the incidence of Malaria. In the Eastern Command the incidence during 1973 was almost three times that of the preceding year. More significant is the gross preponderance of falciparum infection in some areas with atypical clinical presentation and associated complications with a few fatalities. The second problem is one of drug resistance. A few cases of falciparum Malaria showing some degree of resistance to 4-aminoquinolines, which are the mainstay of our prophylactic and curative chemotherapy, have been encountered. The object of this paper is to discuss the diagnosis, complications and management of Malaria in the light of the above.

DIAGNOSIS

2 In a typical case, the diagnosis of Malaria is simple and straightforward. But atypical presentations may pose a diagnostic problem.

(a) **Malaria in immunes living in an endemic area**--The clinical picture does not resemble fully developed Malaria and is modified by immunity. The typical febrile paroxysms are absent. Usually there is malaise, mild fever, headache, anorexia, sweating and a sense of being un-well. More severe attacks occur only if immunity is weakened by inadequate dosage of anti-malarial drugs or if there is loss of immunity due to eradication of infection.

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Troops who have stayed for a long period in an endemic area and have acquired some immunity due to repeated exposure to infection fall in this category.

(i) A clinical profile that was frequently seen in Mizoram was one of mild or moderate short term fever with severe headache and bodyaches and naso-pharyngeal catarrh closely mimicking on acute viral infection.

(ii) In another area a large number of cases were reported with little or no fever but with vague symptoms like malaise, headache, anorexia and gastro-intestinal disturbances.

(iii) Some cases presented with jaundice, hepatomegaly and other symptoms resembling and often mistaken for Viral Hepatitis.

A high index of suspicion was responsible for these cases to be detected. The diagnosis in all these cases was made on the basis of parasitaemia which was sometimes quite marked even in the absence of fever.

(b) **Malaria in Pregnancy**—The clinical picture of Malaria may be modified in pregnancy where it is known to cause abortion or premature labour.

(c) **Malaria in Childhood**—Children, especially in the one to three years age group, are susceptible to malaria and its complications with a fatal outcome in most cases. The clinical picture of acute malaria in a non-immune child differs from that seen in the adult. Fever tends to be moderate. Severe gastro-intestinal symptoms are frequently present. There is often tender hepatosplenomegaly. Convulsions, coma and meningismus are common. Death usually ensues in untreated cases. In chronic cases cachexia, severe anaemia and gross hepatosplenomegaly are prominent features. Icterus and secondary avitaminosis may be present.

(d) **Chemoprophylaxis**—also modifies the clinical presentation. The typical febrile paroxyms with periodicity may be absent when the individual has been on some form of suppression.

(e) **Length of Erythrocytic Cycle of the parasite and mixed infection**—Quartan periodicity of P. malaria, Subtertian periodicity of some strains P. Falciparum and quotidian periodicity of any of the species due to variation in the length of the erythrocytic cycle of the parasite and absence of periodicity in mixed infections as well as in some cases of P. falciparum can cause difficulty in diagnosis.

(f) **Complications**—Also modify the clinical picture and divert one's attention from the primary illness.

3. Complications

Serious complications of Malaria occur primarily in falciparum infection due to impaired capillary circulation, destruction of erythrocytes or toxaemia. Signs and symptoms will depend upon the site of the vessels involved. The complications can be classified as follows depending on the system affected :—

(a) **Nervous system**—55% of all complications involve the nervous system. The commonest is cerebral malaria presenting with acute fever and disturbance of sensorium. Besides this, acute organic mental syndrome where there is disorientation and mental confusion without depression of sensorium, movement disorders like chorea or tremors, focal neurological signs in the forms of transient ischaemic attacks, monoplegia or hemiplegia and acute personality changes may occur. CSF may show moderately increased pressure with pleocytosis or protein increase or both.

(b) **Cardiopulmonary complications**—are mainly in the form of cardiac failure with pulmonary oedema. This in a high altitude area may be mistaken for High Altitude Pulmonary Oedema (HAPO). Rarely acute coronary insufficiency due to obstruction of smaller coronary vessels by parasitised erythrocytes can occur. This was encountered in one of the cases in the Eastern Sector.

(c) **Gastro-intestinal complications**—may produce a picture of acute dysentery or cholera.

(d) **Hepatobiliary complications**—comprise of Bilious remittent fever and Hepatic failure. The latter may be accompanied by renal failure and present as hepato renal syndrome. This was seen in a few of our fatal cases.

(e) **Renal involvement**—may result in an Acute Nephritic picture with renal failure as in P. falciparum infection or in Nephrotic syndrome as in P. malarial infection.

(f) **Haemopoietic**—Blackwater fever where there is acute massive intravascular haemolysis resulting in haemoglobinuria, rapidly developing anaemia, shock and renal failure is well known. Besides, haemolysis may be drug induced as in the case of Glucose-6-Phosphate Dehydrogenase deficiency triggered off by 8-aminoquinolines or phenacetin.

(g) **Reproductive system**—Abortion in malaria of pregnancy has already been referred to.

(h) **Miscellaneous complications**—include—

(i) Algid malaria where there is acute circulatory failure usually without fever and

(ii) Splenic rupture from minor trauma in chronic cases with massive splenomegaly.

4. Management

The management comprises of general and supportive measures and the specific antimalarial chemotherapy. The general management is the same as in any other fever.

(a) The aims of chemotherapy in an acute attack in non-immunes are quite different. In non-immunes the object is eradication of all phases of the infection. In immunes what is required is control of the clinical attack without eradication of the erythrocytic phase of the parasite upon which the immunity depends. The drug of choice is chloroquine of the 4-aminoquinoline group. The standard chemotherapy for an acute attack is as shown below :—

(i) **In Non-immune persons**—Chloroquine diphosphate or sulphate 600 mg base (4 tablets) followed in 6 hours by 300 mg base (2 tablets), then 300 mg base (2 tablets) daily for 2 days. Alternatively 600 mg on the first day, 600 mg on the second day and 300 mg on the third day—Total of 1500 mg—may be given. Amodiaquine dihydrochloride or dihydrate 600 mg base (3 tablets) followed by 400 mg base (2 tablets) daily for 2 days can be also used as an alternative to chloroquine therapy.

(ii) **In immune persons**—A single dose of Chloroquine diphosphate or sulphate 600 mg base (4 tablets) or Amodiaquine dihydrochloride or dihydrate 600 mg base (3 tablets) is often adequate.

(b) **Radical Cure**—The term radical cure refers to eradicating not only the erythrocytic, but also the exoerythrocytic parasites of an established infection as in P. Vivax, P. ovale and P. Malariae.

In the case of P. falciparum where there is no exo-erythrocytic phase, it involves gametocidal action as well as rapid elimination of the primary tissue phase.

This is achieved by the use of Tab. Primaquine 7.5 mg base (3 Tablets of 2.5 mg each) twice daily for 5 days usually along with Chloroquine therapy.

5. Treatment of Pernicious Malaria (P. Falciparum infection with associated complications)

P. Falciparum infection requires early diagnosis and prompt treatment in view of its dreaded complications. 4-aminoquinolines remain the most effective drugs of first choice. Quinine is only indicated when there is evidence of resistance of malaria to 4-aminoquinolines. In a fully conscious patient oral therapy is given as in other forms of malaria. Total dose of 1.5 G over a period of 3 days in non-immunes.

(a) **Cerebral Malaria**—Cerebral malaria must be considered as a possibility in all cases of acute fever with disturbance of sensorium. Blood smear must be repeatedly examined in all such cases. When in doubt it is better to treat the case as cerebral malaria.

Parenteral therapy is indicated when there is high parasitaemia, high temperature combined with shock, diarrhoea or vomiting or any signs indicating hepatic, renal or cerebral dysfunction.

Chloroquine base 200 mg should be administered I.M; half the dose in each buttock initially. This may be repeated at intervals of six hours, but the total dose for the first 24 hours should not exceed the equivalent of 900 mg base. In severe cases and where there is urgency, 600 mg. Chloroquine base can be given I.V in a glucose drip over a period of 30-40 minutes. I.V therapy may precipitate hypotension and must be given slowly. Oral therapy should be resumed as soon as the patient is conscious.

In cases of Chloroquine resistance, Quinine Dihydrochloride 650 mg in Glucose saline or Isotonic saline can be given slowly by I.V drip in about 30 minutes. This may be repeated 6 hourly upto a maximum of 2 G in 24 hours.

In children the parenteral dose of Chloroquine base should be 5 mg/kg body weight. This may be given in two divided doses by I.M injection, with an interval of 6 hours between the doses. In extremely ill children 0.5 mg/kg body weight Chloroquine base I.V in glucose saline can be given over a period of 15 minutes, using a final solution strength not more than 1 mg%.

(b) **Supportive Therapy in Cerebral Malaria**

(i) **Measures to reduce Cerebral Oedema**—Steroids-Inj Dexamethasone 8 mg I. V/I. M Stat and 4 mg I.V/I.M six hourly thereafter for 4-5 days. This helps also to reduce toxaemia and damage to capillary endothelium.

Low molecular weight Dextran (Lomodex)—10-40 mls/ kg body weight on the first day and subsequently 10 mls/kg body weight I.V for next four days. This also acts as a desludging agent and. corrects prerenal uraemia.

Lasix—40-80 mg I.V/I.M as required

(ii) **Anti-convulsant Therapy**—Dilantin sodium 100 mg twice or thrice daily or Diazepam 10 mg I.V every 15 minutes upto a maximum of 60 mg in 2 hours until seizures are controlled in case of recurrent convulsions.

(iii) **Anti-coaqulant Therapy**—I.V Heparin 50 mg (5000 units) may be given six hourly for first 48 hours to prevent clotting of blood in capillaries in comatosed patients. The risk of haemorrhage outweighs its advantages and makes its use debatable.

(c) **Treatment of Black Water Fever**—The principles of therapy in Black Water Fever are mainly directed against anaemia, circulatory failure, dehydration, hepatic failure and renal failure.

If the patient is already on quinine, it should be stopped. Antimalarial treatment with Chloroquine will be required only in the event of substantial parasitaemia, which is rare.

Corticosteroids are helpful in the early stages to prevent further haemolysis.

Blood transfusion is indicated if the erythrocyte count falls quickly below 2 million/in mm, even when haemoglobinuria is absent. Packed cells should be given if there is circulatory embarrassment. Grouping and cross matching should be thorough to avoid blood incompatibility. In the presence of anuria 0.5 to 1 litre of blood is usually adequate. Circulatory overloading should be avoided.

At the same time adequate hydration should be maintained. Hepatic and Renal failure should be treated appropriately when present.

6. **Problem of Drug Resistance**

The emergence of drug resistance strains of malarial parasite in various parts of the world is becoming an increasingly important problem. Proguanil and Pyrimethamine resistant strains have been known for some years. More recently, the emergence of strains of P. Falciparum resistant to 4-aminoquinolines has caused concern and has restricted the usefulness of these compounds in certain areas, particularly in South East Asia and South America. Similar reports of Chloroquine resistant falciparum malaria are available now in India, and some cases have also been encountered in the Eastern Sector amongst our troops.

Amongst other factors, administration of subclinical doses of the drug and irregular or inadequate chemo-prophylaxis, especially if the same drug is used for treatment of the acute attack as well as for suppression, may be responsible for the development of drug resistance.

Cross resistance usually occurs between 4-aminoquinolines and mepacrine. It is also seen between Proguanil and Pyrime-thamine.

Drug resistance may be partial or complete. WHO has defined four levels of response to a standard course of treatment with Chloroquine consisting of 1.5 G of drug base given over a period of three days. The grading of response is primarily based on the behaviour of asexual parasitaemia as shown in the table below:—

Grading of response of P Falciparum to Schizonticidal drug therapy

Response	Recommended symbol	Evidence
Sensitivity	S	Clearance of asexual parasitaemia within 7 days of the first day of treatment without recrudescence
Resistance	R I	Clearance of asexual parasitaemia as in sensitivity, but followed by recrudescence
	R II	Marked reduction of asexual parasitaemia but no clearance
	R III	No marked reduction of asexual parasitaemia

S represents full sensitivity

R I & II represent partial resistance

R III represents total resistance.

The persistence of parasites in the blood does not necessarily mean the persistence of fever. In partially immune persons living in endemic areas the threshold parasitaemia for fever is high and fever may be absent in spite of significant parasitaemia as noted in a number of our cases.

Recrudescence is defined in this context as renewed parasitaemia at a microscopically detectable level within one month from the first day of treatment (day 0). The definition given in "Terminology of malaria and of malaria eradication (WHO 1963, P. 115)", is as follows:—

"Renewed manifestation of infection (Short term relapse) believed due to survival of erythrocytic forms".

Recrudescences sometimes respond to a further course of a standard dose of Chloroquin or to a larger dose of the same drug (eg) 2.4 Gm base, if the resistance is of a very mild degree. Otherwise a different drug or drug combination is necessary.

Almost all infections with Chloroquin resistant strains of P. falciparum can be controlled by the administration of Quinine, given in the adult dosage of 560 mg (10 grains) of the salt three times daily for 10-14 days. If this is followed by recrudescence, the following combinations of drugs may be used :—

(a) Quinine, 650 mg of salt 3 times daily for 14 days, and Pyrimethamine, 50 or 75 mg daily for the first 3 days. Diphenyl sulfone (DDS) 25 mg daily may be added to this regimen with better results.

(b) Several combinations of short and long acting sulphonamides or sulfones with Pyrimethamine have been used for the treatment of acute Chloroquine resistant falciparum malaria. Of the long acting sulphonamides, sulformetoxine has been mostly used either singly or in combination with pyrimethamine. A single dose of 1 Gm of Sulformetoxine with 50 mg of Pyrimethamine has been used extensively with success. A further dose of 0.5 Gm of Sulformetoxine may be given on the second day. A partial answer to the problem of resistance of P falciparum to 4-aminoquimolines has emerged from the drug screening programme of the U. S. Army. Several compounds belonging to 9-phenanthrene methanols and 4-quinoline methanols are reported to have shown high activity on human volunteers.

Resistance of P. Vivax against 4-aminoquimolines and mepacrine has not been reported so far.

Although several cases of Chloroquine resistance have been reported in various areas in the Eastern Sector, only 24 cases have come under my personal observation and are under review. Out of these, 8 cases (33.3%) responded to a subsequent course of Chioroquine—4 required a second course of a standard dose of 1.5 Gm and 4 required a third course of similar dosage. The remaining 16 cases (66.6%) required Quinine—Three of these cases (12.5%) had shown no response whatsoever to chloroquine and had persistent parasitaemia and/or fever indicating total resistance to Chloroquine. Two out of the 16 cases treated with Quinine have had a further recrudescence a month later and have responded to a second course of Quinine. The use of other drug combinations has not been necessary so far.

7. SUMMARY

The difficulties in diagnosis of malaria especially in the presence of atypical clinical presentations and complications have been discussed. The need for high index of suspicion for the early diagnosis and prompt treatment of the pernicious forms of malaria has been stressed. The management of malaria and its important complications has been dealt with in some detail. The problem of drug resistance and the treatment of Chloroquine resistant P. falciparum infection have been discussed briefly.

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CONCLUDING REMARKS OF THE CHAIRMAN

It is vital to recognise that Malaria is again raising its head. The index of suspicion should therefore be high. Recrudesence, development of resistance of the parasite to the anti-malarials and of the vector to the insecticides are serious problems. The clinical state of complicated Malaria is an added hazard. There is consequently need today, more than ever before, for strict vigilance, rigid enforcement of anti-malaria measures and a close-knit team work between different disciplines.

VIRAL HEPATITIS : LABORATORY ASPECTS

BY

Col R.N. Dutta*

INTRODUCTION

The term 'Viral hepatitis' includes two clinical conditions, one caused by type A hepatitis virus and is synonymous with 'infectious hepatitis' and the other caused by type B hepatitis virus which is synonymous with "serum hepatitis". These two diseases have certain clinical, pathological, epidemiological and immunological similarities as differences.

The discovery of Australia antigen by Blumberg et al (1968) and its association with viral hepatitis, type B (Giles et al 1969; Krugman et al, 1970; Prince, 1968) opened up a new vista in the study of natural history of this disease. The lack of suitable laboratory methods for the study of type A hepatitis has obviously hampered progress, but the discovery of Australia antigen has provided at long last, a specific laboratory marker of infection with the virus of type B hepatitis. This antigen under the electron microscope showed it to be of three morphological types consisting of the 20 nm sphere, filaments and the Dane particles of 42 nm sphere (Gerin, 72). There is common antigenic determinants in all the morphologic forms and an additional one peculiar to the Dane particles (Bond, 72). All the three particles reveal the same six polypeptides and an additional one with an estimated molecular weight of about 65,000. (Gerin, 72). The core of the Dane particle is suggested to contain nucleic acid.

The accumulated evidence indicates that the predominant form (20 nm) of HBAg is not itself the virus of HBAg, but rather an incomplete form containing virus specific surface proteins. The virus may be present in much lower concentration. The Dane particle would seem to fit many of the criteria for the virus resembling morphologically the rhino viruses (Zuckerman, 72). Several laboratories have reported success in transmitting the HBAg to sub human primates including chimpanzees and rhesus monkeys Marston, 72; Maynard et al, 72). Recently, success has been claimed in growing Dane particles in tissue culture (Melnick,72).

The tests that are currently available (Table 1) now for the detection of Au-antigen have been shown in the table below.

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Table 1. Current Methods of detection of HBAg.

No.	Types	Sensitivity	Speed	Percentage positive
1.	Immunodiffusion	1	1–3 days	25–60
2.	Counter Immuno electrophoresis	15–20	1–2–3 hours	50–90
3.	Complement fixation	30	1 day	70–95
4.	Haemagglutination Inhibition	30–50	1 day	90–95
5.	Radio immune assay	100–1000	3–6 days	97–99
6.	Latex agglutination	30	Few Mins	90

Ouchterlony technique of gel diffusion lacks sensitivity and detects only 30 percent of cases (Cherubin et al, 1970.) Further, it requires a minimum of 24 hours to give a positive result. Complement fixation method requires 1 to 2 days and is 30 times more sensitive than immuno diffusion. Radio-immune assay (Hollinger et al, 1971) requires about 3 to 6 days but 100 times more sensitive than haemagglutination inhibition and 1000 times more sensitive than immuno diffusion. Equipment, reagents and technical skill required for carrying out this test is not easily available. One of the easiest as well as rapid method is the application of immuno electrophoretic principles to the detection of Australia antigen (Prince, 1970), counter-immuno-electrophoresis (Pesendorfer et al, 1970) and immune electro diffusion by paper discs (Duquesnoy et al, 1970). The counter immuno-electro-phoresis takes about 1 to 2 hours for the precipitation lines to appear and is about 15 to 20 times more sensitive than the immuno diffusion technique.

Another advantage with this technique is that the reagents move in one direction rather than diffusing out radially from the wells so that there is a gain in sensitivity. Recently latex agglutination method (Leach et al 1971) has also been described which is extremely rapid but throws up too many false positive reactions.

Present paper will discuss the laboratory aspects of Viral hepatitis especially the detection of Au-antigen in the laboratory.

MATERIAL & METHOD

Material: 524 cases of Viral hepatitis were investigated for the presence of Australia antigen and antibody. Most of the positive cases referred from local hospitals were tested for Australia antigen every week. Those from distant place could not be followed up so regularly.

To find out the general level of incidence in the local population 923 healthy army personnel, 466 civilian non professional and 730 professional donors were tested for the Au-antigen and antibody.

During the course of investigation of viral hepatitis routine tests like serum bilirubin, thymol turbidity and flocculation, S.G.OT and S.G.P.T. were also carried out.

Method: The following techniques were adopted to investigate Viral hepatitis cases for Au-antigen.

(i) Immuno-diffusion technique :—Au-antigen and antibody in the serum and plasma were tested by Ouchterlony's method with slight modification (Brummelhuis, 1970; Prince et al, 1970). In an agarose gel containing sodium azide (1 in 1000), six wells with a diameter of 3 mm were punched around a central well of 3mm at a distance of 5 mm. Size of the well was reduced to conserve the quantities of serum specially that containing the Au-antibody. Results were read after 48 hours and 72 hours.

(ii) Counter-immuno electrophoresis :—The method employed has been described by Dutta et al (1972). Briefly, 5 ml of warm 0.6 percent agarose was put on 50 mm × 75 mm size glass plate. This 1 mm thick agarose was punched out longitudinally in sets of 3 by means of a cork borer. Distance between the wells was 5 mm. The central wells were filled up with unknown serum and the anodal side wells were filled with Au antibody (anti HBAG) containing sera whilst cathodal side with that containing Au-antigen (HBAG). The system was subjected to a constant voltage of 200 volts for 2 hours. The buffer used was 0.025 M sodium barbitone buffer with a pH 7.6. The Au-antibody containing sera was obtained from multiple transfused patients, standardised against a serum kindly supplied by Prof. J.P. Soulier of Centra National de Transfusion Sanguine, Paris.

RESULTS AND DISCUSSION

Table 2 shows the result of Au-antigen and antibody tests amongst professional and non-professional voluntary blood donors at Delhi. The non-professional blood donors came from civil population mostly students and factory workers and the Army donors were from the local units.

Table 2 : Au-antigen and antibody positive blood amongst Army and Civil blood donors.

Category of Donors	Total No. Tested	Positive for HBAg		Positive for HBAb.	
		Number	Percentage	Number	Percentage
Professional	730	21	2.88	4	0.55
Army Donors	923	24	2.59	0	0
Civilian Donors	466	12	2.57	4	0.85

Level of HBAg incidence amongst professional blood donors, Civilian and Army donors show no statistical significant difference. Similar has been the findings of Sama et al (1973) form All India Medical Institute, Delhi whose findings have been 2.73% amongst professional donors and 2.72% amongst non-professional civilian donors. From this study, the level of HBAg positive sera in general population at Delhi has been found to be rather high as compared to 1% incidence as reported from Bombay by Kulkarni et al (1972). Similar high incidence has been reported by Shanmugham et al (1972) with an incidence of 2.9 percent amongst 239 relative blood donors from Vellore, Tamilnadu.

Table 3 shows the incidence of HBAg amongst 524 cases of viral hepatitis referred to this centre for investigation. Data from reports published by workers are shown for comparative study.

Table 3—Incidence of Au-antigen in cases of Viral hepatitis

Author & Place	Number of cases	HBAg positive	
		Number	Percentage
Dutta R. N. 1972 (Delhi)	524	106	20.23
Mohan T. K. 1973 (Gwalior)	52	16	30.78
Anand and Dutt, 1973 (Poona)	126	42	34.1
Blumberg et al, 1966 (U.S.A.)	77	20	25.9
Okochi et al, 1968 (Tokyo)	167	23	13.7
Prince et al, 1970 (New York)	288	172	59.7
Soulier et al, (Paris)	67	17	25.4

There were 524 cases of viral hepatitis in this series admitted to Defence service hospitals for treatment. Of them, 106 (or 20.23 per cent) showed presence of HBAg. This is rather a low figure as compared to that reported from New York (Prince et al, 1970) and Mohan (1973) from Gwalior as shown in table 2 above. In the present study, observations on the presence of HBAg in the pre-icteric phase have rather been limited. Not all the 524 cases in this study could be followed up every week, as many of them were referred from hospitals outside Delhi. 63 local cases, which could be followed up every week were tested for serum bilirubin, S.G.O.T., S.G.P.T. other liver function tests and also for Australia antigen and antibody.

Fig. 1 and 2 show that during the icteric phase, S.G.O.T. and S.G.P.T. levels were raised in all cases which were positive for HBAg. Transaminase level came down to normal nearly immediately after the serum bilirubin level came down to normal and this usually occurred on an average, in 4 to 6 weeks of time. The Australia antigen, similarly remained positive usually for a period of 4 to 6 weeks. However, there were a few cases in this series where the Australia antigen persisted for a period longer than 5 weeks and in one case it is still persisting for more than 48 weeks leading to a chronic antigen carrier state. It has been reported by Redeker (1972) that unresolved viral hepatitis can occur in 10 to 12 percent of icteric hepatitis cases. It has also been stated by the same author that there is a close relationship between unresolved viral hepatitis and persistence of HBAg and that 1% of bio-chemically healed patients demonstrated presistent antigenemia.

The factors which may lead to the progression of acute hepatitis to chronic liver disease are (1) initial hepatitis to be HBAg positive and (2) the disease is mild or anicteric (Alter, 1972). This is characteristically seen in one of the cases who was admitted on 15.3.73 for Jaundice which cleared up within a month. His SGOT/SGPT levels which in the beginning were raised to 82 and 128 respectively came down to the normal level within a month, but he has consistently been positive for HBAg even today after a period of 48 weeks. The seriousness of anicteric hepatitis and its progression to subacute hepatic necrosis and post necrotic cirrhosis has been well documented by Dr. Klatskin (1958). Sherlock et al (1970) has described 17 cases who were HBAg positive, chronic liver disease. Ten of them had no past history of acute viral hepatitis. Vogel et al (1972) demonstrated HBAg in 40 percent of patients with hepatoma superimposed on post-necrotic cirrhosis ; none had a history of previous hepatitis. Baker and Murray (1971) demonstrated by volunteer studies that persistent carriers of HBAg had mild anicteric hepatitis or totally inapparent disease and all these people had received highly dilute or modified HBAg positive plasma.

Such asymptomatic apparantly healthy HBAg carrier is typically exemplified in a Sep/BTA of my own unit who, when volunteered to donate blood during 1972, was

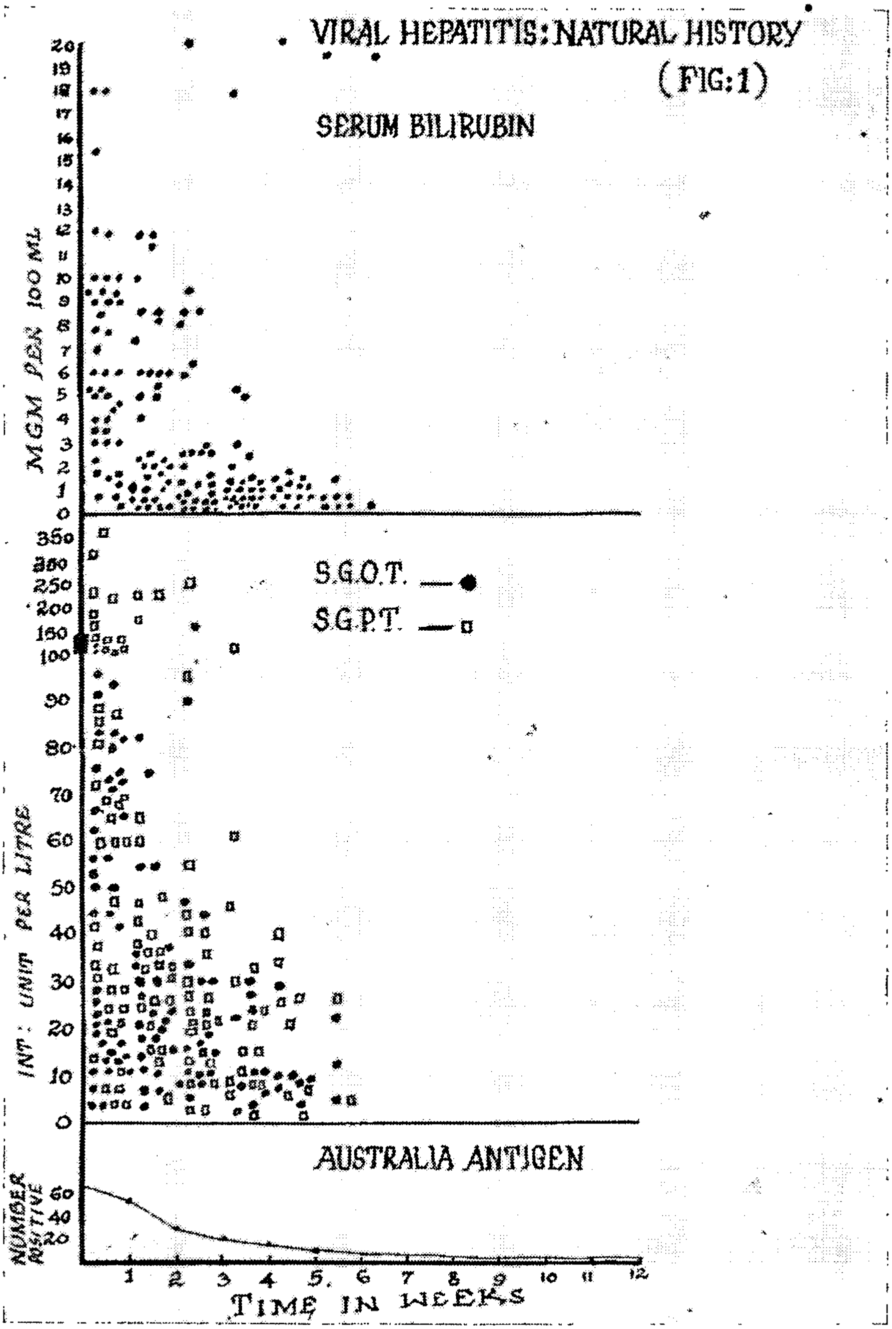

Fig. 1—Coorelation of HB Ag with S. B., SGOT/SGPT

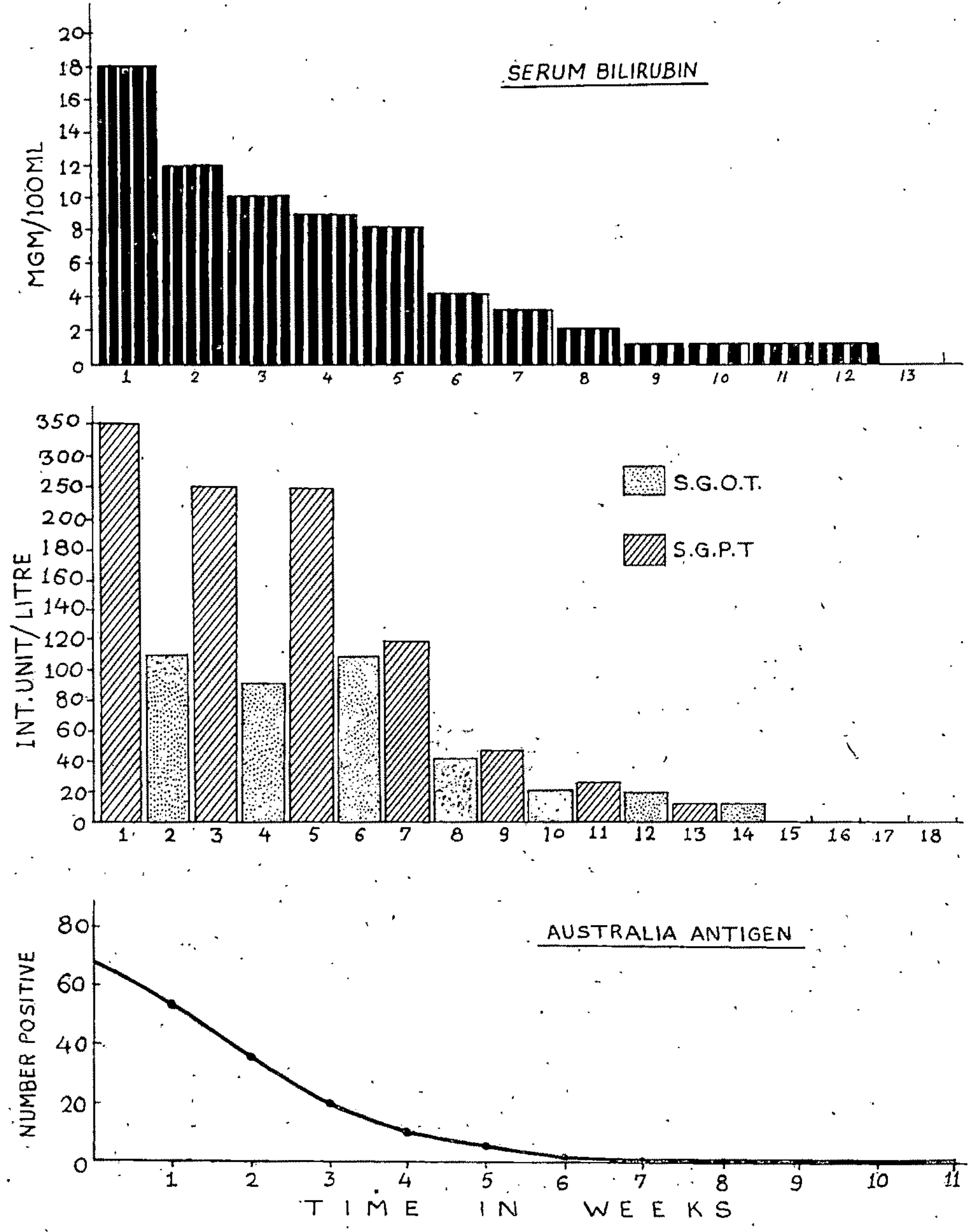

Fig. 2—Correlation of HB Ag with S. B., SGOT/SGPT

found to be strongly positive for HBAg and is still strongly positive today with normal serum bilirubin, SGOT and SGPT levels. Such anicteric asymptomatic cases may have a more serious long term implications than the acute clinical disease.

Okochi and Murakami (1968) first reported the occurrence of viral hepatitis after transfusion of HBAg positive blood. HBAg was detected 4 to 16 weeks after receiving HBAg positive blood transfusion. According to Blumberg et al (1971) 69% of recipients of blood containing HBAg develop viral hepatitis but the author does not state whether all of these cases became positive for HBAg subsequently.

Post transfusion hepatitis is exemplified in the case of mitral valvotomy (fig. 3) who was transfused with HBAg positive blood. HBAg became positive on the 50th day after transfusion, when the SGOT and SGPT level started rising. The transminase level went upto 98 and 40 units/litre on the 80th day after transfusion when the icterus became obvious. HBAg remained positive till 130 days when the SGOT/SGPT level came down to normal with the disappearance of the icterus. Of the 524 Viral hepatitis cases, only in 4 cases Australia antibody could be detected. Two of the anti-HBAg positive cases did not show HBAg whilst the other two cases had both HBAg and anti-HBAg.

Levene and Blumberg (1969) first described the multiple antigenic specificities of Au-antigen. It is now known that HBAg has a group antigen 'a' which is shared by all HBAg positive sera and two subsidiary specificities called 'd' and 'y' which are not equally shared by all such sera (Le Bouvier, 1972). These two antigens are mutually exclusive, virus specific and not host-dependent (Le Bouvier, 1971). HBAg positive sera could thus be grouped as D (ad+dy—), Y (ay+d—) or A (a+dy—).

64 of the HBAg positive viral hepatitis cases were subtyped with subtype specific guinea pig antisera ad and ay kindly supplied by Dr. R. Byrne of National Institute of Allergy and Infectious diseases, National Institute of Health, Bethesda, U.S.A. Table 4 below shows the results of the subtyping of these 64 HBAg positive sera and compare the same with the cases at Copenhagen (Nielsen et al, 1973). 11 healthy carriers were also subtyped which is also shown in the same table.

Table 4 : Au-antigen subtypes among viral Hepatitis cases and Healthy carriers.

Place	No. of Patients	Y Subtype	D Subtype	Percentage D Subtype
Copenhagen (Nielsen et al, 1973),	183	145	33	21
Dutta (Delhi, 1974)	64	45	16	25
Healthy Carrier (Copenhagen)	23	0	23	100
Healthy Carrier (Delhi)	11	8	3	27

Both at Cophenhagen and at Delhi, the Y subtype was found to be predominant subtype in viral hepatitis cases, whereas, D subtype was 100 percent in healthy carriers at Copenhagen as opposed to only 27 percent at Delhi. But the number of healthy carriers tested at Delhi are so few that one would be hesitant to draw any conclusion from such a small series. Nielsen et al (1973) have also shown that 98 percent of the drug addicts had the Y subtype whereas D subtype predominated in the sporadic cases. It has been also stated by the same authors that D subtype had a more severe disease than Y subtype. This aspect also needs to be studied in our country.

SUMMARY

923 healthy army personnel 466 healthy non-professional and 730 professional blood donors were tested for HBAg to find out its incidence in the general population at Delhi. The incidence was 2.59, 2.57 and 2.88 percent respectively. Serum from 524 cases of viral hepatitis was tested for HBAg and 20.23 percent of them were found positive for HBAg.

Only 63 of these cases could be followed up regularly at weekly intervals for serum bilirubin and other liver function tests, SGOT, SGPT and HBAg. With the disappearance of Jaundice in most cases, SGOT, SGPT levels came down to normal along with a negative test for HBAg. Occasional cases showed exceptions and few became persistent carrier of HBAg. One anicteric asymptomatic carrier is described with implications and future seriousness of such a situation.

64 HBAg positive viral hepatitis cases and 11 healthy carriers were subtyped, 45 of the patients and 8 of the healthy carriers were found to be of subtype Y and 16 of the patients and 3 of the healthy carriers were found to be of D subtype.

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VIRAL HEPATITIS—CLINICAL ASPECTS

BY

Lt Col R.B. NANDA*

Viral hepatitis is a disease with a worldwide distribution. The average incidence in troops during the past 10 years has been 6 per 1000.

The Committee[1] on hepatitis of the U.S. National Academy of Science, National Research Council classifies viral hepatitis into viral hepatitis type A and viral hepatitis type B. The specific diagnosis is dependent on the presence of hepatitis B antigen in viral hepatitis type B. Designation recommended by the committee in connection with these two types is as follows:-

Disease	*Virus*	*Antigen*	*Antibody*
Viral hepatitis type A	HAV	—	—
Viral hepatitis type B	HBV	HB Ag	HB Ab

Hepatitis type A virus has not been isolated although transmission experiments have shown its presence in blood and faeces in acute phase of disease and in faeces during convalescence. Recently an antigen which appears to be associated with viral hepatitis type A has been detected in a Milanese patient. Zukerman[2] believes that this antigen is probably an abnormal serum lipoprotein which appears as a result of liver damage. Ferris[3] has also described an antigen which is commonly found in the faeces of patients with viral hepatitis type A but rarely in controls. Its role in the disease, however, requires further investigations. Should the Milan antigen prove to be pathogenic it will confirm the oral faecal route of transmission of the disease.

CLINICAL ASPECTS

In general there is little difference between the clinical course of viral hepatitis type A and type B. Diagnostic features that help to distinguish viral hepatitis type A and type B are given below:—

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(a) **Incubation period**— Though variable depending on the dose and route of infection, it is relatively short for type A i.e. 15-50 days (mean 30-35 days). For type B the range is 43-180 days (mean 60-90 days).

(b) **Type of onset**— It is generally acute for type A and insidious for type B. In the latter group it may extend for 2–3 weeks and may be associated with serum sickness like symptoms marked by arthritis involving distal joints, fever, urticarial rash and angioneurotic oedema. This syndrome has been attributed to circulating immune complexes of HB Ag, HB Ab and the complement. The levels of C_3 component are very low in these patients.

(c) **Clinical features**

(i) During prodromal period fever is common in type A and is infrequent and milder in type B.

(ii) The severity of the type B is very variable. The mildest attack may be asymptomatic. Transient antigenaemia and a rise in transminase levels may be the only clues to the diagnosis. Alternatively the patient may still be anicteric but suffer gastrointestinal and influenza-like symptoms. These types are likely to remain unrecognised unless a patient gives history of needle puncture or transfusion.

(d) **Mortality**—Type B has a higher mortality. In type A the mortality may be 2 per 1000 whereas in type B the mortality can be high as 1 in 8 for the post-transfusion patients and 1 in 200 for the naturally acquired disease.

EXTENT OF VIRAL HEPATITIS B IN THE ARMED FORCES

We are all conversant with viral hepatitis type A but probably not with proved cases of viral hepatitis type B. Our experience of type B at Base Hospital, Delhi Cantt is described in the succeeding paragraphs.

Incidence

Out of 202 caases of viral hepatitis admitted in Base Hospital, Delhi Cantt during the period March 72 to October 73, 52 were of viral hepatitis B.

Predisposing Factors

(a) **Age**—The age of the patients varied from 14–50 years. The mean age was 28.2 years. The maximum number of cases were between 21 and 30 years (Table 1).

Table 1. Age Distribution

Age (years)	No. of cases
11—20	5
21—30	37
31—40	7
41—50	3

(b) **Injections/Tranfusion**—There was history of TAB inoculation in 8, vaccination in 4, combined TAB inoculation/vaccination in 10, combined TAB/TT in 3 and combined TAB/vaccination and TT in 2 cases during the preceding six months (Table 2). History of blood transfusion was available in 6. In all 33 cases had some sort of needle puncture. The maximum incidence was during the first three months (Fig. 1).

VIRAL HEPATITS—CLINICAL ASPECTS

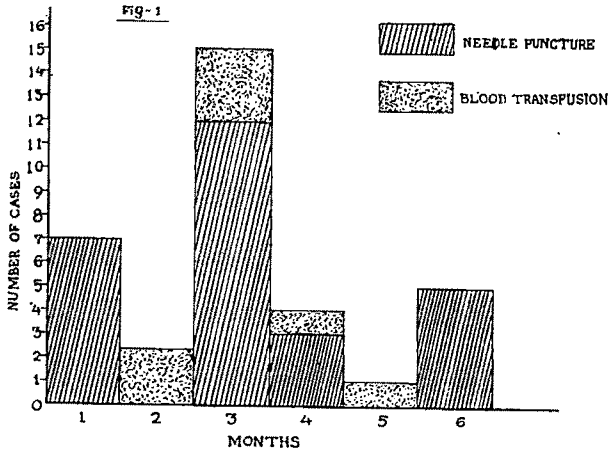

Fig. 1 . Relation ship needle puncture during preceding six months.

Table 2. Relationship with Needle Puncture during preceding six months

	No. of cases	Months					
		1	2	3	4	5	6
TAB	8	–	–	6	–	–	2
Vaccination	4	2	–	–	–	–	2
TAB+Vacc	10	4	–	4	2	–	–
TAB+TT	3	1	–	1	1	–	–
TAB+Vac+TT	2	–	–	1	–	1	–
Blood Transfusion	6	–	2	3	1	–	–

Clinical Features

The common clinical features were anorexia, nausea, pain epigastric/right hypochondrium and arthralgia. Fever was present in 12 cases. Diarrhoea, pruritis and dysosmia were observed in a few cases (Table 3).

Table 3. Clinical Featurers in 52 Cases

Symptoms	No. of cases	Percentage
Anorexia	45	87
Nausea	34	65
Pain Rt Hypochondrium	26	50
Arthralgia	23	44
Vomiting	17	32
Fever	12	23
Diarrhoea	9	17
Pruritis	4	7
Dysosmia	1	2

Duration of persistence of HB Ag/HB Ab

The persistence of antigen in the blood was found to last from 1–10 weeks. In one case who developed chronic hepatitis, antigen persisted for 22 weeks. In 2 cases antigen is still persisting (even after 44 weeks in one case and 15 weeks in the other). The range of persistence of antibody was 2-4 weeks. In 2 cases HB Ab was complexed with HB Ag and persisted for 2 and 3 weeks respectively (Table 4). The presence of antibody or antigen antibody complex did not affect the course and severity of disease.

Table 4. Duration of persistence of Hb Ag/HB Ab

Duration (weeks)	HB Ag	HB Ab	HB Ag/HB Ab complex
1-2	9	–	1
3–4	12	2	1
5–6	13	–	–
7–8	7	–	–
9–10	4	–	–
* 15	1	–	–
22	1	–	–
* 44	1	–	–

* Still persisting

Relationship between antigenaemia and size of liver enlargement

Antigenaemia per se had no relationship with the size of liver. Thus in 5 cases liver was not palpable although antigenaemia persisted for 46 days. On the other hand in 8 cases where the liver was palpable by 3 cm, HB Ag returned to normal in 39 days (Table 5).

Table 5. Relationship between Antigenaemia and the size of Liver

Size of liver	No. of cases	HB Ag returned to normal in (days)	Liver size returned to normal in (days)
Not palpable	5	46	—
Upto 1 cm	18	38	29
Upto 2 cm	18	52	74
Upto 3 cm	8	39	62
Upto 4 cm	2	38	42
Upto 5 cm	1	46	85

Relationship between Antigenaemia and duration of liver enlargement

There was no correlation observed between the duration of Antigenaemia and liver enlargement (Table 6).

Table 6. Relationship between Antigenaemia and liver enlargement

Duration of liver enlargement	No. of cases	HB Ag returned to normal (days)
Upto 2 weeks	8	33
Upto 3 weeks	5	62
Upto 4 weeks	7	34
Upto 5 weeks	3	40
Upto 6 weeks	5	47
Upto 7 weeks	5	72
Upto 8 weeks	3	52
Upto 9 weeks	1	14
Upto 12 weeks	2	50
Upto 14 weeks	1	38

Incidence of positive HB Ag among contacts

In the Army Forces as most of the personnel live in barracks, there is every chance of transmission of hepatitis B. virus from person to person. In contacts the initial attack may be mild and may not be associated with jaundice. The contacts can become asymptomatic carriers of HB Ag and if not detected in time can be a source of infection to others. To determine the extent of the problem 55 contacts of 17 HB Ag positive cases were studied. The following abnormalities were found in these contacts :—

(a) HB Ag was detected in 3 contacts (5.4%). It persisted for 4, 8 and 16 weeks respectively. Only the third case showed cellular infiltration and fibrosis in the portal tract on liver biopsy.

(b) HB Ab was detected in one contact. It persisted for 2 weeks. We thus have a high incidence of HB Ag positive cases amongst our troops. These observations, however, need to be carried out on a wider scale.

Chronic Antigen Carrier

The HB Ag is usually cleared in 3–4 weeks. If it persists for more than 12 weeks after the onset of illness, the individual is likely to become a chronic carrier. In 52 cases studied in this series antigen is persisting even after 44 weeks in one and 15 weeks in another. In both these cases liver biopsy showed no evidence of chronic hepatitis. The disposal of carriers in the Armed Forces poses a practical problem. It seems unrealistic to isolate or restrict the activities of most long term HB Ag carriers. They should be advised of the need for meticulous hand washing, of the inherent danger of donating blood and of sharing of razors or tooth brushes.

MANAGEMENT

Acute Viral Hepatitis

There are no revolutionary advances in the treatment of acute viral hepatitis. As the course of the disease is unpredictable, it is better to treat all cases with bed rest and high protein diet. They can be allowed up when they are asymptomatic. They may be sent on sick leave when the liver is no longer tender and the serum bilirubin is less than 1.0 mg per 100 ml.

Corticosteroids are not indicated in typical acute viral hepatitis. They neither slow the rate of liver cell necrosis nor increase the rate of liver cell regeneration. Many patients with chronic sequelae of hepatitis and persistent antigenaemia have received steroids during the acute attack. Steroids do not prevent the development of chronic hepatitis. They are, however, beneficial in patients with prolonged cholestasis and in those passing into subacute stage with persistent jaundice.

Tetracyclines are toxic to liver and they can lead to hepatic dysfunction, azotaemia and pancreatitis. Their use in the presence of hepatitis is, therefore, contraindicated.

Fulminant Hepatic Failure

Hepatic coma is a dreaded complication of acute viral hepatitis. The recovery rate at present is not more than 10 per cent. Its early recognition is, therefore, of paramount importance.

Measures should be adopted to prevent the formation of ammonia in the gut. Diet should have minimum of 1600 calories in the form of glucose drinks or 20% glucose intravenously. All proteins should be excluded from the diet. Neomycin 1-1.5 mg 6 hourly should be given. Lactisyn 1 ampoule orally every 4 hours alters the intestinal flora. Magnesium sulphate 15-30 mg should be given orally and bowel wash should be carried out once or twice daily.

Recent Trends in management of Fulminant Hepatic Failure

The measures should be directed towards stimulating the regeneration of liver cells either spontaneously or therapeutically. But currently available methods do not influence the regeneration. Exchange blood transfusion, plasmapheresis and hyperbaric oxygen are no longer popular.

Beneficial role of isolated pig liver perfusion has not still been established as marked thrombocytopaenia and fall of antibodies are big hazards. The role of corticosteroids is controversial. No benefit has been observed. They are known to perpetuate infection and peptic ulceration.

L–dopa was used with the hope that it will increase the concentration of dopamine and nor–epinephrine in the brain. Although it improves the level of consciousness with associated improvement of electro encephalographic appearances, the final outcome is still unsatisfactory. Liver transplantation despite the enormous advances in surgical technique and immunopathology has also not proved helpful.

Haemorrhagic diathesis due to intravascular coagulation and impaired hepatic synthesis of coagulation factors aggravates hepatocellular failure. Treatment to prevent haemorrhages should start even before evidence of bleeding. Heparin is given 4000 units is bollus dose. Further dosage is adjusted by estimating the plasma heparin level.

Recently heperimmune serum globulin containing HB Ab has been used with success in the treatment of HB Ab positive cases[4].

Finally, haemoperfusion (artificial liver) technique in which blood is perfused through charcoal to remove water soluble toxins and through ion exchange resins to remove protein bound compound has been developed. So far two patients treated by hemoperfusion have survived. Preliminary results are promising.

SUMMARY

Two hundred and two cases of viral hepatitis admitted in Base Hospital, Delhi Cantt were studied. The overall incidence of viral hepatitis type B was 25.8%. Thirty-three cases (63.2 %) had TAB innoculation, vaccination, tetanus toxoid injections or blood transfusion during the preceding six months. Arthralgia was prominent feature in 44% of the cases. Mild to moderate fever was present in 23%. Characteristic serum sickness-like syndrome described in the Western literature was not encountered in our cases. Liver was not enlarged in 5 cases (9.6%). No correlation between liver enlargement and persistence of antigenaemia was noticed. Incidence of HB Ag amongst 55 contacts of 17 viral hepatitis type B was 5.4 per cent (3 cases). In the contacts, antigenaemia lasted for 4 weeks, 8 weeks and 16 weeks respectively.

In 2 cases (1.9%) HB Ag is still persisting, after 44 weeks in one and 15 weeks in another. The carriers if not detected early would be potential source of infection to

others. Role of corticosteroids, exchange blood transfusion, hepatic transplantation, L–dopa, heparin, hyper–immune serum and haemoperfusion has been discussed.

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VIRAL HEPATITIS IN THE ARMED FORCES—EPIDEMIOLOGIC ASPECTS

BY

Lt Col R. N. Varma*

INTRODUCTION

Perhaps, second only to malaria and scrub typhus, Viral Hepatitis almost wholly the "infectious" form constitutes an important health hazard for troops particularly in field areas, and invariably ranking first during peace times. It has been a scourage of fighting armies and has, therefore, been aptly designated as the "Jaundice of Campaigns". The disease produces disabling illness resulting in man-power wastage which is largely preventable.

This communication presents a broad epidemiological picture of "Viral Hepatitis" in the Armed Forces against the background of current international thinking on the subject. However, at the outset it must be admitted that though, we have witnessed several revolutionary advances during recent years towards a better understanding of this disease, it still remains an important unresolved problem in medicine.

EPIDEMIOLOGIC FEATURES

Definition—The term "Viral Hepatitis" connotes two diseases that are clinically quite similar but epidemiologically, immunologically, and virologically distinct; these diseases are "infectious hepatitis" and "serum hepatitis" now known as Viral Hepatitis type A and Viral Hepatitis type B respectively. Any other viral infection that affects the liver producing an inflammatory response or hepatitis is not normally included under the term "Viral Hepatitis". It is difficult to differentiate between individual cases of Hepatitis A and Hepatitis B based on clinical picture alone, but it is possible to do so from a detailed evaluation of the epidemiologic picture and blood tests for Hepatitis-B.

Terminology—A multitude of terms have come into vogue for the two diseases over the past years; as many as 21 synonyms are used for Hepatitis-A and 28 for Hepatitis-B (World Health Organisation, 1973). The more commonly known synonyms are shown in Table I below :—

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Table I

SYNONYMS FOR VIRAL HEPATITIS

Viral Hepatitis Type A	Viral Hepatitis Type B
Acute Catarrhal Jaundice	Australia Antigen Hepatitis
A-IH Hepatitis	Hepatitis B (H.B.)
Epidemic Catarrhal Jaundice	Homologous Serum Hepatitis (Virus-B)
Hepatitis A (H.A.)	Long-Incubation Hepatitis
MS-1 Hepatitis	MS-2 Hepatitis
Short-incubation Hepatitis	Serum Hepatitis (S.H.)
Viral Hepatitis Type A	Viral Hepatitis Type B

The disease is often acquired as a result of a particular type of exposure and terms such as "syringe or needle associated", "Chimpanzee associated", "transfusion associated" and "Haemodialysis associated" help specify the particular mode of transmission. Until recently, the two forms of the disease were distinguished on long held precepts of route of infection, the period of incubation and on the results of studies of transmission in human volunteers. But, the discovery of Australia antigen in 1963 and its subsequent confirmation in 1967 as a specific indicator of infection with hepatitis-B placed the distinction between these two diseases on a firm foundation (World Health Organisation, 1973). The disease is now referred to as :—

Viral Hepatitis Type A and

Viral Hepatitis Type B with its associated Hepatitis-B antigen and its corresponding antibody.

The variations in the important epidemiologic, clinical and biochemical features of hepatitis A and hepatitis B are summarised in Table II below :—

Table II

FEATURES OF "INFECTIOUS" AND "SERUM" HEPATITIS

Feature	"Infectious Hepatitis"	"Serum Hepatitis"
Epidemiologic		
Route of infection	Mainly Fecal-Oral but also Parenteral	Mainly parenteral but also fecal-oral
Seasonal incidence	Autumn and winter	All Year

Feature	"Infectious Hepatitis"	"Serum Hepatitis"
Age distribution	Children/young adults	All ages
Duration of infectivity and carrier State	Blood: for days Feces: for weeks to months	Blood: for months to years Feces: Undetermined
Ratio of icteric to anicteric	About 1 : 10 in adults About 1 : 2 in children	About 1 : 100
Mortality Rate	0.1% to 0.3% in young ; Higher in older individuals especially in women after menopause	Upto 1%
Clinical		
Onset	Usually abrupt	Usually insidious
Admission to hospital	Early	Later in the disease
Anorexia	Present	Usually absent
Abdominal pain	Infrequent	Frequently appearing after 1 to 2 weeks
Ambulation	Usually nonambulant	Usually ambulant
Arthralgia	Usually insignificant or absent	Common in Pre-icteric phase ; worse at night
Fever (38°C)	Usually common	Less common
Biochemical		
Prophylactic value of Gamma Globulin	Good	Variable results
SGOT	Spiking short lived rise (1-3 weeks)	Gradual and prolonged rise (upto 3 months)
Thymol Turbidity	Abnormal	Relatively normal
IgM	High increase 3-4 days before abnormal SGOT, returns to normal 5-35 days later.	Slight incerease during acute stage
Chlorination		
Filtration and Chlorination	Effective	Not done
Chlorination alone	Less effective	Not done

Viral Hepatitis Type A—Viral Hepatitis-A, though principally transmitted by the fecal–oral route under poor sanitary conditions and close contact with infected persons is now also known to be spread by the parenteral and possibly other routes. The infection is also present in susceptible sub-human primates and, therefore, poses an occupational risk to persons coming in contact with such animals. Administration of Gamma Globulin prevents or modifies an attack. The virus has not yet been grown in any cell or organ system and, therefore, the possibility of active immunisation is yet to be explored. There are no specific tests for the disease and no specific treatment. Morbidity is variable and mortality low. Viral Hepatitis Type B:—Exposure to virus infected blood and its products by the parenteral route alone does not explain the recent increase in the worldwide incidence of the disease. We have witnessed a marked shift in the age and sex specific rates for the malady in some of the advanced countries of the world, the preponderance of cases in the age group 15-29 years, their clustering in urban areas and the levelling off of seasonal peaks have been found to be associated with the ever increasing abuse of illicit drugs and the peculiar mode of life of drug addicts with greater chances of non-parenteral transmission. The ingestion of blood or inhalation of blood aerosols also plays part in transmission. Amounts as small as 0.0004 ml of plasma can transmit the disease. In the tropics ritual circumcision, tattooing and scarification are additional risks. Biological and mechanical transmission by arthropods particularly *culicine* mosquitoes has also been postulated, venereal spread is also suspected; and transplacental and perinatal transmission from the mother to the infant have also been suggested as an important means of maintaining the infection in the community (World Health Organisation, 1973). These unusual modes of spread if or when confirmed will add new dimensions to the proper understanding, prevention and control of this baffling disease. Administration of Gamma Globulin gives variable results in preventing or attenuating an attack. Active immunisation with inactivated vaccine (MS-2 Serum) has been tried but it needs further evaluation in the field, to prove its value.

Prince (1971) described the preparation of a special immune globulin from plasma containing HB Ab that had a titre of 50,000 to 100,000 times higher than standard immune globulin. Using this preparation Krugman (1971) reported on its efficacy in the prevention of type B hepatitis by passive immunization. Of 10 susceptible children who received infectious serum containing HBV and were inoculated 4 hours later with special immune globulin, six had no disease and 1 had transient HB Ag. All 11 controls who were not protected developed characteristic HB infection.

Problem in the Armed Forces

The reported incidenee of "Viral Hepatitis" in the Armed Forces applies mostly to "Infectious Hepatitis" as it is classified under *infective and parasitic diseases* [1] (070), Serum Hepatitis being included as a complication of medical procedures in the section on injuries,

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1. The current International Statistical Classification of Diseases, Injuries and Causes of Death—Vol. I, 1967 W.H.O., Geneva.

according to nature of injury (N-Code) and according to external causes (E-Code). Because of this differentiation in the reporting system, the difficulty in pinpointing serum hepatitis clinically and practically non-availability of specific laboratory aids[2] for hepatitis-B antigen, the peripheral hospitals and other medical establishments normally report all cases of "hepatitis" as that of the "infectious" variety.

The annual incidence of the disease during the last two decades is indicated in Figure 1 below, which shows that the disease has been showing a continuing rising trend during the period 1952-71; the incidence which stood at 3.81 per thousand during 1952 rose to 4.59 during 1961 and to 5.80 in 1971. The years 1956 and 1966 witnessed the peak incidence at 6.36 per thousand and 13.47 per thousand respectively; in 1956 it was the result of an explosive water borne epidemic in Delhi in December 1955 and in 1966 the rise was due to the 'Siliguri' out-break which resulted from sewage pollution of water supplies at the Siliguri Railway Station and was partly the result of spill over from the unusually high incidence during the Kutch operations of 1965.

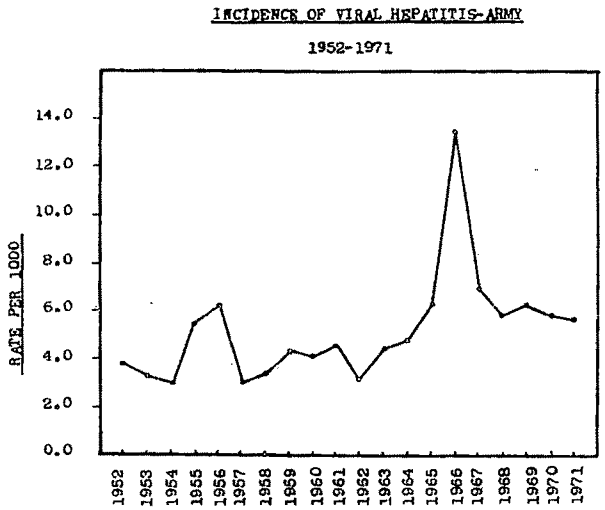

Fig. 1—The incidence of the disease during the last two decades.

In marked contrast to the rising incidence of infectious hepatitist during the decade 1952-71, the incidence of other excremental diseases viz. the Diarrhoea-Dysentery complex registered a significant decline (Figure 2) during the same period particularly in the latter part of the decade 1962-71. It is reasonable to attribute this decline to the overall improvement that has steadily taken place in environmental sanitation,

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[2] The various techniques for measuring Hepatitis-B antigen and anti-body are given in WHO Technical Report Series No.512-1973.

Fig. 2

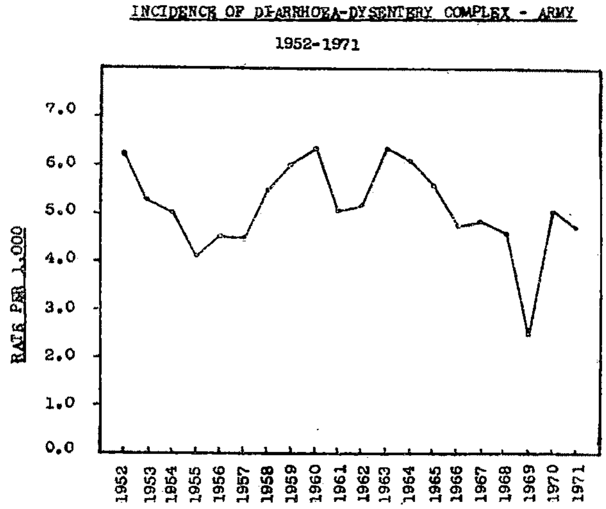

Fig. 2—Incidence of Diarrhoea-Dysentery Complex in Army during 1952-1971.

gradual provisioning of water borne latrines in place of the conservancy system, fly proofing of kitchens and dining halls in new construction and above all to effective health education, both personal and impersonal, of troops and their families in matters of personal hygiene. In the face of this overall improvement in hygiene and sanitation this differential in morbidity due to "infectious hepatitis" and the "Diarrhoea-Dysentery complex" is rather intriguing. But, this could be explained by (a) the "endemic prevalence of the disease all the year round in the country and (b) in the majority of cases the infection seems to have been acquired by troops from consumption of food and water from unauthorised sources, when they are away from the disciplined life of the Unit while in transit either on leave or temporary duty. Contaminated water supply at Railway Station is also an important culprit.

The incidence of the disease Command-wise (1963-72), which is depicted in Figure 3, shows that it is more or less equally distributed in the Southern, Eastern, Western and Central Commands**. The average monthly incidence of the disease during the period 1966-72 is shown in Figure 4, from which it is clearly discernible that the disease is prevalent all through the year, the peak in a particular month being due to sporadic localised outbreaks.

** Northern Command came into existence in 1972 only.

Fiq. 3

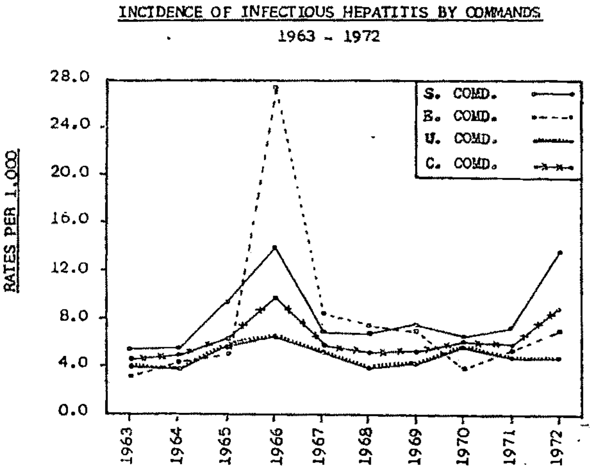

Fig. 3—Incidence of infectiou Hepatitis by commands durings 1963–1973.

Fiq. 4

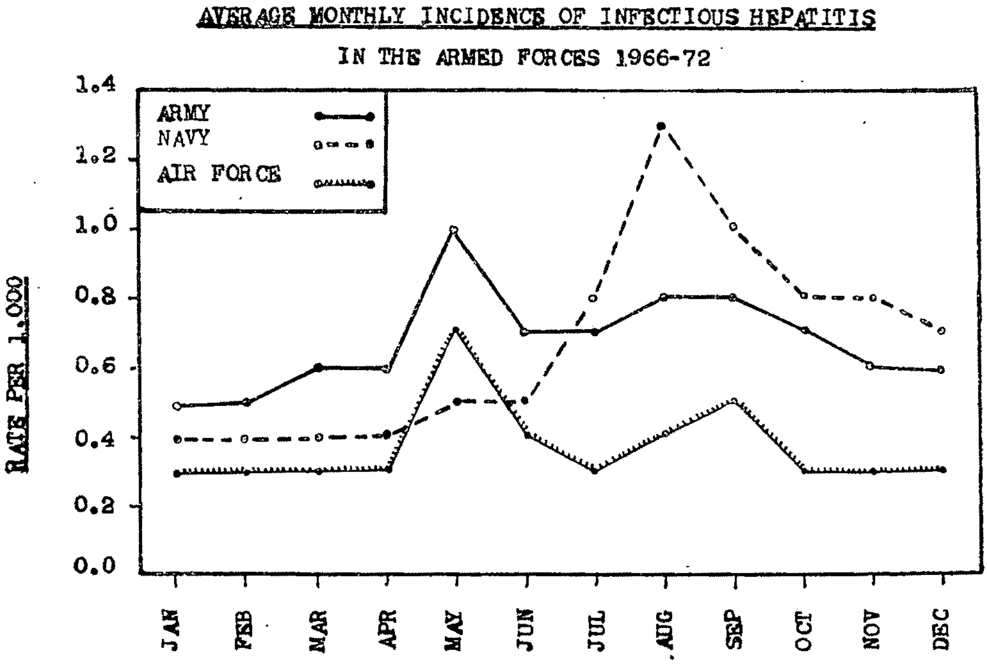

Fig. 4—Average monthly incidence of infectious Hepatitis in the Armed Forces during 1966–72.

The incidence of Infectious Hepatitis in the Army, Navy and the Air Force for the years 1963-72 is shown in Table III below :—

Table III

Statistical Significance of the year to year variation in the Incidence of Infectious Hepatitis in the Armed Forces 1963-72.

Year	Army	Navy	Air Force
1963	4.49	2.98	2.53
1964	4.79 NS	5.53 S	2.25 NS
1965	6.42 S	5.50 NS	4.19 S
1966	13.47 S	6 42 NS	6.09 S
1967	7.00 S	5.30 NS	3.96 S
1968	5.90 S	5.73 NS	5.60 S
1969	6.23 S	13.66 S	4.77 S
1970	5.90 S	4.97 S	4.78 NS
1971	5.79 NS	6.33 S	3.75 S
1972	7.30 S	9.17 S	4.34 S

S=Significant

NS=Not Significant

Note : The level of significance was fixed at the difference between the two rates being more than twice its standard error.

It is seen from Table III that the disease has recorded a rise not only in the Army but the Navy and the Air Force have also experienced higher incidence during the same period. The peak incidence at 13.66 per thousand in the Navy during 1969 was due to polluted water supply from the Bombay Municipal Corporation during the latter half of the year.

DISCUSSION

The incidence of 'Infectious Hepatitis' in the Armed Forces has registered a rising trend during the past two decades and to-day it leads the group of excremental diseases for hospital admissions. Nearly 4500 cases are admitted each year to Service Hospitals with an average incidence during the past decade at around 6 per thousand with a mortality of 2 per thousand. The average duration of stay in the hospital of a case of infectious hepatitis is about 30 days followed by 8 weeks of sick leave. The cost in terms of man days lost due to hospitalisation and sick leave each year is, therefore, very substantial and is a big drain on trained man power. Not a year has passed in recent times without sizeable outbreaks in some place or the other. The epidemic of infectious hepatitis in Delhi during 1955-56 (Sanyal, 1957), the Bombay outbreak of 1958-59 (Sharma, 1959) and the Kirkee outbreak of 1966 (Verma and Ganguli 1969) have all been well documented and there are many more episodes of more or less equal magnitude which have not found their way in the literature and are confined to official records and reports only.

The disease is endemic all through the year throughout the length and breadth of the country. It has, however, not received due attention from the civil public health authorities. Its incidence therefore, in the civil population cannot even be estimated and figures are not available either for the country as a whole or for any state.

Troops and their families have to cast their lot with their civilian compatriots and, therefore, naturally share the burden of morbidity and mortality from this leisurely moving and smouldering disease. There are two main culprits in this regard (a) the total dependency of troops and their families on water supplies under civilian control in big cities like Delhi, Bombay, Calcutta, Madras, Lucknow, Pune etc. It needs no emphasis that all the ills of failure, break-down, inadequacy and contamination necessarily go with such sources of water supply. (b) Water supplies at Railway Stations, constitute the other notorious source from which the infection is contracted by troops when they are on the move. Here availability of clean water, is more often than not a real and acute problem operating almost all the year round. It is, therefore, easy to account for the fairly heavy toll which 'Infectious Hepatitis' inflicts on troops in Garrison Stations and while on train journeys compared to which the incidence under field service conditions is generally of a lower magnitude.

Even in military cantonments the water supply is not always free from blame. For some time past it has not been possible to ensure provision of safe drinking water at all times to troops due to non-availability of chlorine gas cylinders for want of repair/overhaul of chloronomes at some stations. The alternative of manual chlorination with bleaching powder is no substitute for automatic gas chlorination and this has been partly responsible for the mounting incidence of infectious hepatitis amongst troops and families during recent years. However, this problem is receiving serious attention.

The usual modes of spread of Infectious Hepatitis are either a common vehicle spread or from person to person spread. The common source outbreaks usually explosive in nature are of serious import and account for a majority of such episodes in the services. The common source spread may be a single exposure or prolonged exposure over a period of time in which case the epidemic curve becomes widened. The epidemic in the Rajasthan Sector in 1971-72 provided the classical example of person to person spread resulting from insanitary conditions particularly unhygienic disposal of night-soil. This was one instance where attention to field hygiene could have avoided the episode

Prevention obviously assumes added significance in the Armed Forces to avoid man power wastage. The common preventive measures which are all very well known need no repetition except for an emphasis on the following ones :—

(i) Effective health education of troops to change their outlook towards this disease so that when they are away from the disciplined life on the Unit they maintain self discipline and do not eat or drink from unauthorised sources.

(ii) Good personal, group and community hygiene.

(iii) Effective environmental sanitation procedures.

(iv) Quality control and constant monitoring of water supplies to avoid possible contamination and ensuring gas chlorination of properly clarified and filtered water with 1.00 ppm of residual chlorine.

The true incidence of "Serum Hepatitis" in the Armed Forces is not known although some cases do come to light from a cross check of fatal documents and notification forms. Some cases give a history of inoculation/vaccination 160 days (the upper limit of incubation period of Serum Hepatitis) prior of onset of the disease but no definite relationship between the two is established. Its prevention depends on good hospital and public health practice, ensuring a separate syringe and needle for each prophylactic or therapeutic injection and excluding from blood donors the carriers of Hepatitis-B antigen and preferably having no more than five donors in a pool.

SUMMARY

A broad epidemiological picture of viral hepatitis in the Armed Forces bringing out its potential impact on man power wastage is presented. This disease heads the group of excremental diseases for hospital admissions accounting for nearly 4,500 cases each year. The disease registered a rising trend during the past two decades in sharp contrast to the declining trend in the incidence of dysentery and other diarrhoeal diseases during the same period. An effort has been made to explain this differential. The role of effective health education in personal and community hygiene and provision of potable

water in the prevention of the disease is highlighted. The latest global thinking on the disease is briefly reviewed.

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CONCLUDING REMARKS OF THE CHAIRMAN

The nomenclature and clinical picture of Viral Hepatitis are clearer today. Sixty to seventy per cent of the incidence is attributable to infection with Virus A. A major portion of this incidence can be eliminated through health education and observance of principles of personal hygiene and sanitation. As 20 to 30% of the cases are due to type B Virus, the necessity for adequate sterilisation of syringes and instruments is obvious. The role of Australia Antigen and other discoveries in the field have opened up a fascinating chapter in clinical medicine.

AMOEBIASIS—MEDICAL ASPECTS

BY

Surgeon Captain G. Kuppuswamy, VSM

INTRODUCTION

Amoebiasis covers a wide range of diseases produced by infectious by pathogenic amoebae primarily due to *entamoeba histolytica.* The disease is of worldwide prevalence particularly so in tropical regions. It is endemic in our country and is of frequent occurrence in our ARMED FORCES. The incidence reported is variable, i.e. 3.8 to 47.4 per cent.

The following table gives the Prevalence of Intestinal Amoebiasis :—

Author	**Region**	**Incidence**
Tribedi & De (1938)	Calcutta	10%
Chaudhuri & Rai Chaudhuri (1946)	Calcutta	8.8%
Gupta et al (1954)	Eastern India	11.1%
Cherian et al (1954)		7%
Suesson (1959)	Nepal & West Bengal	11%
Wats et al (1958)	South India (Secunderabad)	10.6%
Subramaniam et al (1954)	South India	38%
Chhuttani et al (1961)	North India	13.6%
Dubey et al (1965)	Delhi	9.7%
Mathews (1956)	Lucknow (UP)	3.8%
Anand B.R. (1954)	Rajasthan	19.7%
Amin et al (1962)	Baroda	10.3%
Patel J.C. (1945)	Bombay	43.5%
Srivastava J.B. (1953)	Bombay	20.1%
Pacheco De Fiqueiredo (1959)	Goa	7.1—16.2%
Grewal (1968)	Delhi	47.4%
G. Kuppuswamy (1973) (unpublished)	ATNK & K Area	4.87%

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Incidence of Amoebiasis in ATNK & K Area

Amoebiasis (all types)	4.87%
Intestinal	0.9%
Intestinal and Hepatic	2.6%
Non-suppurative hepatitis	1.2%
Hepatic Abscess	0.17%

The reported variation in the incidence is due to :

1. The type of population examined, whether asymptomatic general population, patients with general abdominal symptoms, those with diarrhoea or patients having dysentery.
2. Differences in the size of the sample
3. Variable techniques of examination
4. Competence of the technician.

The diagnosis and treatment of amoebiasis is a very satisfying experience to a clinician but very many times due to its ubiquitous presentation it baffles and humiliates the best of physicians. The irregular course, chronicity, frequent relapses and remissions, wide variety of intestinal and visceral complications, absence of any definite relevant history and lastly but importantly the feature of the total negativity of the usual investigations carried out, in many cases makes it a unique and perplexing clinical entity. On many occasions it is only a therapeutic response that will establish the diagnosis in retrospect.

Experience with this disease in the ATNK & K Areas during Jan. 1972-73 is presented. For uniformity and clarity, the WHO classification detailed below has been used.

W.H.O. (1969) Classification of Amoebiasis

Asymptomatic
Symptomatic

Intestinal Amoebiasis

Dysentery
Non-dysenteric Colitis
Amoeboma
Amoebic Appendicitis

Complications and Sequelae of Intestinal Amoebiasis
Perforation and Peritonitis, Haemorrhage
Intussusception, Post Dysenteric Colitis, Stricture

Extra Intestinal Amoebiasis

Hepatic

(1) Acute Non-suppurative
(2) Liver Abscess

Complications of Liver Abscess

Rupture or extension, bacterial infection.

Haematogenous spread to other organs

Cutaneous
Involvement of other organs i.e. Lung,
Brain, Spleen etc. without manifest Liver
involvement.

Intestinal Amoebiasis

In most people Intestinal Amoebiasis causes little or no clinical illness but in certain circumstances it may cause catastrophic illness with serious morbidity and mortality. The clinical presentation is protean and no single symptom or sign is pathognomonic of disease as such. The reasons for this extremely variable expression of infection by the same parasite are as yet not clearly defined but many factors are implicated and of these, malnutrition may be a major determinant of morbidity. Differences in the composition of the dominant types of gut flora may also have a bearing on pathogenicity.[19]

By far the commonest mode of presentation is with mild diarrhoea, which may be chronic or relapsing and is unassociated with any significant constitutional disturbance. The stools may be just loose but characteristically there is some excess of mucus. Other vague complaints like abdominal distension and discomfort without real pain "Feeling Windy", tendency for the bowels to open soon after meals, bulky stools suggestive of steatorrhoea and even constipation and vague constitutional symptoms like undue fatigue, low grade fever, vague bodyaches, arthralgia may be present. The dysenteric type is usually charaterised by sudden onset with pain in abdomen, which may be colicky, tenesmus and frequent passage of unformed faeces containing blood and mucus, but fever and constitutional disturbances may not be evident. At times the clinical presentation may mimic an irritable bowel syndrome. Intestinal amoebiasis may co-exist with ulcerative colitis.

Physical examination may not reveal anything significant apart from tenderness along the length of the colon and some thickening particularly of the caecum and pelvic colon. Co-existence of amoeboma may cause diagnostic confusion with granulomata like tuberculosis and even malignancy.

Relevant data from the records of ATNK & K Areas are presented below :—

Agewise Distribution

Age	**No**	**%**
1—10 Yrs	Nil	—
10—20 ,,	11	3.2
21—30 ,,	108	49.0
31—40 ,,	105	30.0
41—50 ,,	18	5.5
51—60 ,,	30	9.0
Above 60 ,,	6	2.6
	341	100 %

The maximal incidence had been between 21–40 yrs probably due to a preselection of the cases. No case was noted between 1–10, this finding is not in conformity with other published reports. Although we treat a fairly large number of patients above 60 years the incidence of amoebiasis is limited in this age group.

Duration of Symptoms

Duration	**No**	**%**
Less than 1 month	78	23
1—6 months	38	10.6
6—12 months	18	5.5
1—2 years	12	3.5
2—3 years	18	5.5

Symptoms

Symptoms	No	%
Dysentery	6	1.75
Diarrhoea	12	3.5
Abdominal Pain		
Generalised diffuse	52	9.4
Lower abdominal Right iliac fossa	13	3.8
Left lower quadrant	9	2.65
Upper abdominal Epigastric	14	4.1
Right hypochondrium	65	19.1
Excessive Flatulance	19	5.6
Diminished Appetite	38	17
Nausea	45	13.2
Anxiety manifestation	9	2.65
Failure to thrive	45	13.2
Fever	58	17

By and large all patients complained of abdominal pain generalised, and localised as indicated above. It is pertinent to note that 19% of patients complained of Rt. hypochondriac pain.

Physical Signs

Physical Sign	No	%
Tenderness lower abdomen	84	26.7
Caecum and Ascending Colon Palpable and thickened	78	25
Tender	101	30
Descending Colon and sigmoid —Palpable and thickened.	125	36.1
Tender	130	53.2
Enlarged Liver Non-tender	26	7.6
Tender	182	53.5

It is of interest to note that in the primarily intestinal form of the disease non tender hepatic enlargement was present in 7.6% and tender enlargement of 53.5% suggesting that active hepatic involvement in intestinal amoebiasis is extraordinarily common. This feature raises the question whether, the conditions can be dissociated at all.

Diagnosis of Intestinal Amoebiasis

The diagnosis of Intestinal Amoebiasis depends on proper history taking, with particular reference to previous diarrhoeal or dysenteric episode, periodicity remissions and relapses and history suggestive of latent visceral complications. This is aided by a meticulous physical examination to elicit signs already mentioned. The following are the various tests that could be carried out in amoebiasis.

1. **Examination of Stools**
    - (a) Macroscopic appearance
    - (b) Microscopic examination
    - (c) Demonstration of *E. Histolytica*—Vegetative forms or cysts.
2. Demonstration of the colonic lesions by sigmoidoscopy.
3. Serological tests
4. Rectal and Colonic Mucosal Biopsy
5. Clinical response to Anti-Amoebic Therapy.

Serology in Amoebiasis

1. Indirect Haemaglutination test.
2. Complement fixation test.
3. Gel. diffusion Precipitation test.
4. Immunoelectrophoresis test for Amoebiasis.
5. Latex Agglutination test
6. Amoeba Immobilisation test.
7. Intradermal test.
8. Fluorescent antibody test.

Repeated careful examination of stool for vegetative and cystic forms of *E. histolytica* must be carried out in all cases of suspected amoebiasis. The percentage of positivity is low but more careful examination of the stool may prove a larger number of cases. Sigmoidoscopic examination is also necessary and should be carried out as frequently as possible in an appropriate phase of the patient's illness.

Rectal and colonic biopsy are indicated where an ulceraivet or other forms of colitis are suspected. The various serological tests are supplementary to establishment of the diagnosis of amoebiasis. In 40 to 50% of cases they are reported to be positive-indicative of invasive amoebiasis.

So far we are not yet carrying out these serological tests in most service hospitals. Even after such detailed investigations a fair number of cases may elude positive diagnosis. In such cases on therapeutic response to anti-amoebic therapy the diagnosis would depend.

The following case illustrates a rare form of phlegmonous amoebic colitis.

A sepoy aged 29 years, admitted as a case of Acute Pharyngitis in one of the peripheral hospitals, developed acute pain in abdomen with frequent watery motions containing mucus and high fever. Clinical examination besides thickened and tender sigmoid colon and palpable caecum did not reveal any significant abnormality. Stool examination revealed presence of pre-cystic *E. histolytica.* A diagnosis of acute amoebic dysentery was made and patient treated with metranidazole (800mg x 8 hrly), terramycin and chlorostrep, and later (after 48 hrs) Dehydroemetine was added to the regimen and I.V. fluids together with other supportive treatment were continued. The condition, however, continued to deteriorate and he became toxic, frequent motions now containing blood and mucus, persisted and he developed distension of abdomen. Clinically a mass extending from R.I.F. to right hypochondrium, exquisitely tender, with muscle guarding developed. Antiamoebic treatment with supportive I.V. fluids were continued but the patient's condition deteriorated relentlessly and he expired about 5 days after developing acute dysenteric manifestations.

Post mortem revealed evidence of extensive amoebic ulceration, necrosis and haemorrhages in the large gut mucosa. Large number of trophozite form of *E. histolytica* were also seen in the depths of the tissue. This case illustrates a rather rare form of fulminant amoebic colitis which ended fatally in spite of vigorous treatment with dehydroemetine, flagyl and broad spectrum antibiotics. The cause of death in this case was colonic ulceration and haemorrhage.

Hepatic Amoebiasis—This includes

(a) Acute Non-Suppurative hepatic amoebiasis

(b) Amoebic Abscess of Liver.

Acute Non-Suppurative Hepatic Amoebiasis

This is a controversial entity. One school of thought believes it to be a pre-suppurative involvement of the liver associated with intestinal amoebiasis in 50% of cases. It is claimed that diffuse amoebic hepatitis without abscess formation without demonstrable *E. histolytica* in liver biopsies is a distinct clinical and pathological entity. Others believe that since no *E. histolytica* are demonstrable in this state the only acceptable lesion of hepatic amoebiasis is the abscess. However, this is a constant and pressing problem in the Armed Forces—the patient presenting with minimal hepatic enlargement —slightly to moderately tender associated with a mild leucocytosis. Most of these patients do not show *E. histolytica* ova or cysts in the stools, the sigmoidoscopy is normal and so is the hepatic scan. In a minority of cases there may be a minimal increase in the serum bilirubin level mimicking infectious hepatitis. Where facilities are available the B.S.P. excretion and alkaline phosphtase levels may be abnormal. Serological studies are reported to be positive—suggestive of invasive amoebiasis in 45.7 per cent of cases. Liver biopsy has been reported to be non-contributory. This is clearly a situation where it is difficult to withhold anti-amoebic therapy particularly when the experience of most clinicians the world over is that this condition responds very well to anti-amoebic therapy. More detailed investigations of such cases should be carried out to elucidate this clinical entity further. Are we over treating such patients ? Would the hepatic enlargement and other symptoms subside by mere symptomatic therapy? If so, in what time? Would the man hours lost be increased by not treating them? Will they develop an abscess if the anti-amoebic drugs are withheld? It will be appreciated how difficult these questions are.

Amoebic Liver Abscess

In the tropics this complication of amoebiasis occurs in about 2 to 10% of cases. It is reported that the carrier state of *E. histolytica* and inadequate anti-amoebic treatment of intestinal amoebiasis are important predisposing causes. Despite the large prevalence of amoebiasis it is not clear why only a few develop liver abscess. Factors like virulence of infection, resistance of host, excessive alcohol consumption, diabetes mellitus and malnutrition may play important roles in rendering the liver vulnerable to abscess formation.

Clinical Features

Classical clinical features of pain, fever, hepatomegaly with palpable abscess are well known. Various authors have, however, laid stress on the atypical presentation of amoebic liver abscess in absence of pain or fever, prolonged unexplained pyrexia, non-tender and firm enlargement of liver and spleen and presence of deep jaundice. It is necessary to emphasise that jaundice is not an uncommon sign in hepatic abscess as thought earlier. Minimal splenic enlargement, minimal ascites and jaundice are red herrings which one must be wary of. Amoebic abscess may be co-existent in an already enlarged and congested liver, e.g. in chronic Rt. heart failure of corpulmonale.

Jaundice in Amoebic Abscess may be due to—

1. Peri and Intra-ductal inflammation.
2. Pressure of abscess cavity on bile ducts distorting intra hepatic biliary tree-intra-hepatic cholestasis.
3. Extensive and diffuse liver involvement due to generalised liver injury or a manifestation of hyper immune reaction to antigen introduced by amoebic infection.

Diagnosis of Hepatic Amoebiasis

1. Examination of stool for cyst of *E. histolytica*
2. Examination of blood
3. Liver function tests
4. Radiology
5. Liver scan
6. Serological tests
7. Diagnostic aspiration

Leucocytosis is usual, the liver function tests may not be substantially altered but where jaundice is present the serum bilirubin level may be raised. In recent times liver scan has taken a lead over radiological examination in the early detection of abscess of liver. Sizeable liver abscess may exist without much radiological evidence of raised rt. dome of diaphragm and fluroscopy may not reveal any limitation of movement of the diaphragm on the right side. Of course diagnostic aspirations clinch the diagnosis. The following case illustrates a case of liver abscess presenting with jaundice.

Case No. 2

A 27 years old Naval Officer was admitted with complaints of high fever and pain right hypochondrium of about 2 weeks duration. He gave a past history of dysentery and was habituated to alcohol. Clinical examination revealed evidence of icterus and mildly enlarged and tender liver. A clinical diagnosis of amoebic hepatitis was made and patient treated as such. Laboratory investigations revealed a polymorphonuclear leucocytosis (total count, 14,500 cm) with poly 78%, raised serum bilirubin (3 mg% of direct reacting type), raised alkaline phosphatase (35 KA units) and Serum transminase (74 Karmen units). X-ray chest (Fig. 1) revealed that slightly raised right dome of diaphragm with normal movements or fluroscopy. In view of the above features a diagnosis of acute cholangitis was considered by the Surgeon and patient transferred to Air Force Hospital, Bangalore for further review. Liver scan (Fig. 2) revealed a large filling defect in the right lobe of the liver and subsequent aspiration drew about 500 ml of typical anchovy sauce pus. With continuance of antiamoebic treatment and repeated aspirations the patient made a total recovery (fig 3 and 4).

This case emphasises that

(a) Jaundice is not an uncommon sign in amoebic liver abscess.

(b) Laboratory evidence of obstructive jaundice with elevated alkaline phosphatase commonly occurs in such cases.

(c) Upward enlargement of Liver is not always seen in cases of amoebic liver abscess.

Pleuro-Pulmonary Amoebiasis

The incidence of Pleuro-Pulmonary amoebiasis has been variously reported between 5% to 15% by various workers.

Pleuro-pulmonary amoebiasis may be :

(i) Primary—in the absence of manifest hepatic lesion.

(ii) Secondary—to manifest hepatic lesion

The secondary spread of amoebic infection from the liver could result from :

(1) Rupture of the liver abscess into pleura or lung.

(2) Transdiaphragmatic spread through the lymphatics

(3) Embolisation of amoebic trophozoites through blood vessels resulting in lesions in mid or upper zones or in multiple lesions.

(4) Left sided involvement through affection of left lobe.

(5) Sympathetic pleural effusion in response to subdiaphragmatic amoebic lesion.

De Bakey classified pleuro-pulmonary amoebiasis into 5 groups as under :

(1) Hematogenous pulmonary abscess without liver involvement.

(2) Hematogenous pulmonary abscess and independent liver abscess.

(3) Pulmonary abscess extending from the liver abscess.

(4) Bronchopleural fistula with little pulmonary involvement.

(5) Empyema extending from a liver abscess.

Most of the cases of pulmonary amoebiasis are missed as they mimic very closely pulmonary tuberculosis. But if one gives attention to the characteristic features of the

Case No. 2

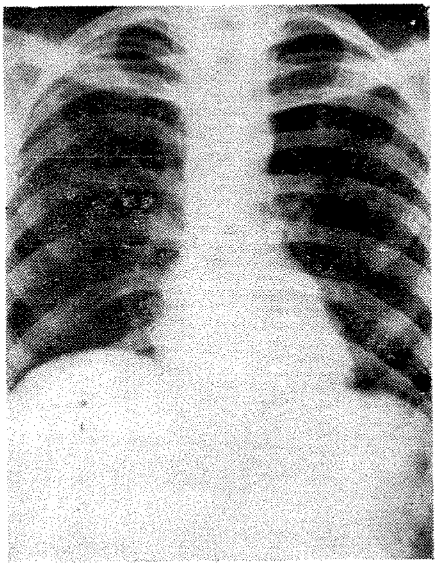

Fig. 1

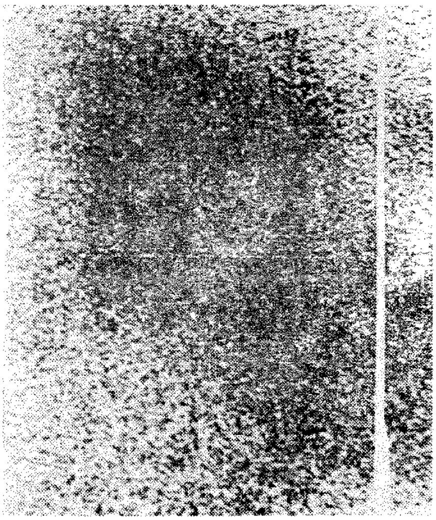

Fig. 2

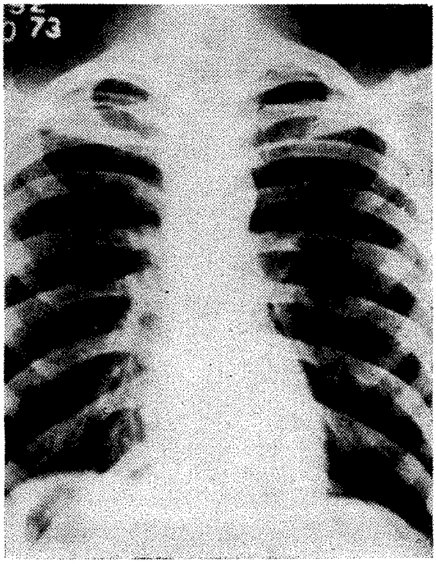

Fig. 3

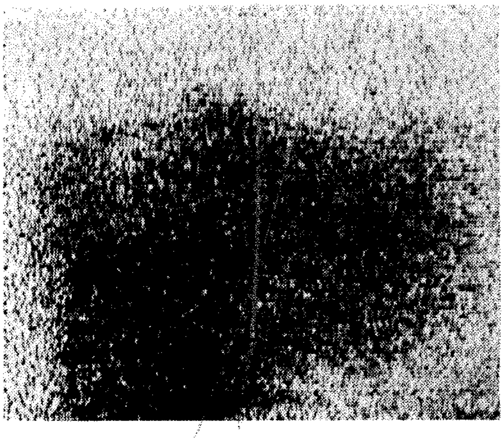

Fig. 4

Case No. 2—Amoebic Liver abscess with Jaundice.

Case No. 3

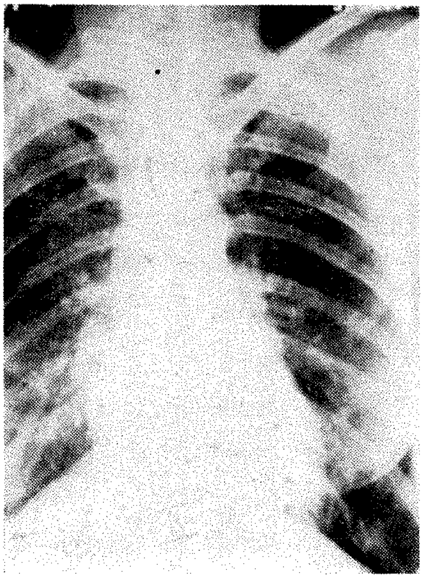

Fig. 5

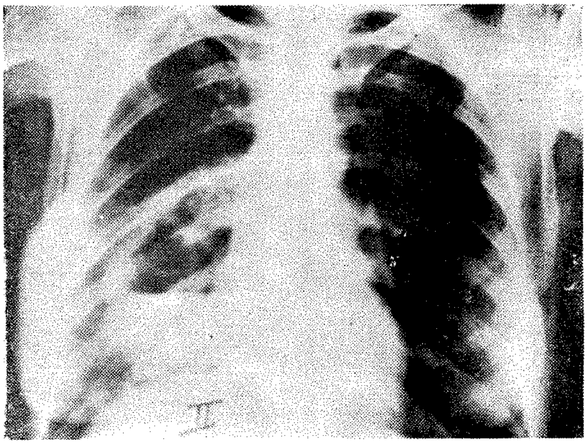

Fig. 6

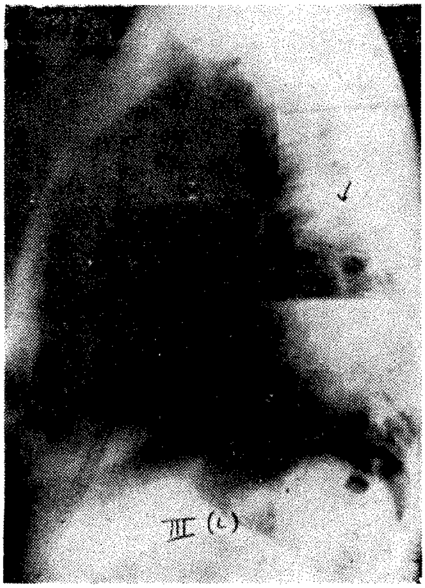

Fig. 7

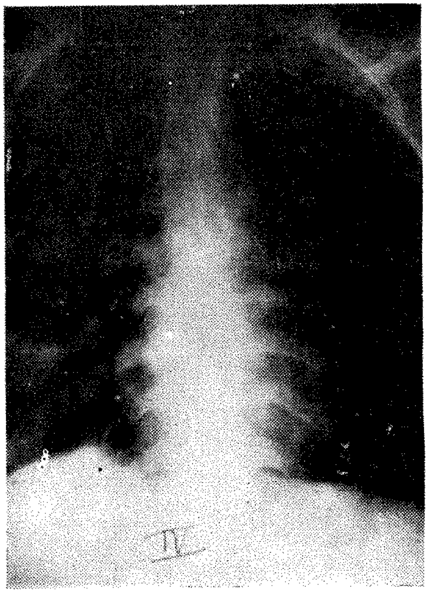

Fig. 8

Case No. 3—Pleuro-pulmonary amoebiasis.

sputum (Brownish in colour) and the presence of associated tender liver, the diagnosis is unlikely to be missed. A strong suspicion and awareness that such a condition exists also play their part.

The following case illustrates pleuropulmonary amoebiasis.

Case No. 3

A 33 year old lady was admitted to one af the peripheral hospitals with complaints of breathlessness, fever, cough with yellowish expectoration and pain in the right lower chest of about 2 days duration. Clinical examination revealed her to be dyspneic with evidence of mild toxaemia. Lungs revealed clinical evidence of pneumonitis over right mid and lower zones (Fig. 5). Sputum was negative for AFB and she was treated as a case of pneumonitis with penicillin bronchodilators and other supportive therapy. She did not show any improvement. Repeat clinical and radiological examination of chest revealed large abscess cavity right lower lobe with fluid and air (Fig 6 & 7). Liver at this stage was slightly enlarged and tender. Stools exam. for *E. histolytica* was negative and sputum culture grew pyogenic cocci. Suitable antibiotics and postural drainage were instituted but she did not show any significant improvement. About 2 weeks later she brought out chocolate coloured sputum. Although careful examination of sputum did not show *E. histolytica* she was put on dehyroemetine and parenteral chloroquin to which she responded dramatically within 24 hrs of institution of therapy. Subsequently X-ray chest taken 3 months later showed evidence of residual fibrosis only with complete resolution of cavity (Fig. 8).

This case manifested pulmonary amoebiasis (i) without significant hepatic involvement and the colour of sputum gave a clue to the diagnosis which was confirmed by dramatic therapeutic response.

Primary Amoebic Meningo-Encephalitis—is one of the rarer manifestations of amoebiasis due to amoebae of the genus Harmannella (Acanthamoeba). Unlike the parasitic amoeba, *E. histolytica,* which may occasionally cause meningo cerebral infection by metastatic spread, these free living amoebae gain primary entrance to the Central Nervous system through the nasopharynx. The term primary Meningo-encephalitis was coined by Butt (1966) for the primary involvement of CNS by the free-living amoebae thus differentiating it from metastatic meningo-cerebral infection due to *E. histolytica.*

Clinical Features

These comprise upper respiratory tract infection and signs of meningeal irritation and mental confusion. Focal or generalised fits and deep coma may ensue.

L.P. shows raised CSF pressure, cells counts are low, protein and sugar are raised. Presence of mobile amoebae clinch the diagnosis.

Sulphanamides and amphotercin-B are effective in therapy.

Asymptomatic Cyst-Passer

A fair percentage of individuals who are totally asymptomatic may persistently pass amoebic cysts. These individuals seem to live in peaceful co-existence with their amoebae. Given an adverse circumstance, however, they may develop serious forms of the disease. From the epidemiological point of view the asymptomatic cyst passer must be fully treated.

Management of Amoebiasis

There have been no dramatic changes in the management of amoebiasis in the recent times. When non contraindicated emetine still remains the drug of choice.

The other drugs are

1. Chloroquine—alternative to emetine in hepatic amoebiasis.
2. Diloxanide furoate—used primarily in the Intestinal amoebiasis and in the treatment of cyst passers.
3. Metronidazole—alternative to emetine in mild to moderately severe cases ; both in hepatic and the intestinal forms.

The usual dosages of these drugs in the average adult are—

1. Emetine Hydrocholoride—60 mgs. deep subcutaneous injection for 6—10 days.

1B. Dehydroemetine—60—90 mgs. deep subcutaneous injection for 6 to 10 days.

2. Chloroquine—300 mgs. twice daily for 21 days.
3. Diloxanide furoate—500 mgs 3 times a day for 10 days—21 days in cyst passer.
4. Mentronidazole 400—800 mgs. 3 times a day for 5—10 days.

(The large single dose therapy recommended in amoebic abscess has been disappointing in my experience). It is necessary for clarity to mention therapy for different forms of amoebiasis.

1. **Symptomless Amoebiasis**—(Asymptomatic cyst passers) Metronidazole for 5 days ; or Diloxanamide furoate for 21 days.

2. **Acute amoebic dysentery**—Metronidazole for 5 days combined with Tetracycline or Oxytetracycline 250 mgs 4 times a day for 5 days; or
   (a) Inj. Dehydrometine or Emetine Hydrochloride for 6—10 days, either continuously or with a break of 3 days in middle of the course.
   (b) Concurrently Diloxanamide furoate for 10 days with tetracycline for 5 days.

It is essential to note that Peritonitis, localised or generalised may develop in Acute amoebic dysentery. In such cases apart from specific treatment, attention should be paid to fluid and electrolyte balance, surgical drainage and exhibition of suitable anti-biotics. Surgical treatment directed towards closing the perforation is not advisable.

3. **Chronic Intestinal Amoebiasis**

(a) Treat for as acute amoebic dysentery.
(b) If symptoms persist after a parasitic cure look for alternative diagnosis such as irritable colon syndrome. Also rule out malignancy.

4. **Amoeboma**

Treatment is same as for Acute Dysentery.

5. **Hepatic Amoebiasis**

(a) Emetine hydrochloride/dehydroemetine for 6—10 days; and
(b) Chloroquine—21 days; and
(c) Diloxanide furoate for 10 days.

OR

(a) Metronidazole for 5—10 days.

If the liver abscess is secondarily infected a course of appropriate antibiotics should be given based on sensitivity tests.

Conclusion

In conclusion the following have to be re-emphasised :—

1. On the slightest suspicion of amoebiasis the case should be thoroughly investigated to confirm the diagnosis and later fully treated.

2. Positive diagnosis may elude the clinician in many cases but the case may

demand anti-amoebic therapy solely on clinical grounds. Therapy in such cases must be exhibited.

3. It is absolutely mandatory, on the slightest suspicion of a liver abscess, that the case should be treated in closest possible liaison with the Surgeon and the Radiologist.
4. Always beware of the elusive presentation of amoebiasis.

Acknowledgement

I wish to thank all the Medical Specialists of the TNK & K Area for their co-operation in the preparation of this paper, the ADMS TNK & K Area for permission to obtain data from the records and the O.C. Air Force Hospital, Bangalore for permission to publish the case reports. I am grateful to Major General R.S. Hoon, AVSM, Senior Consultant in Medicine, for his guidance.

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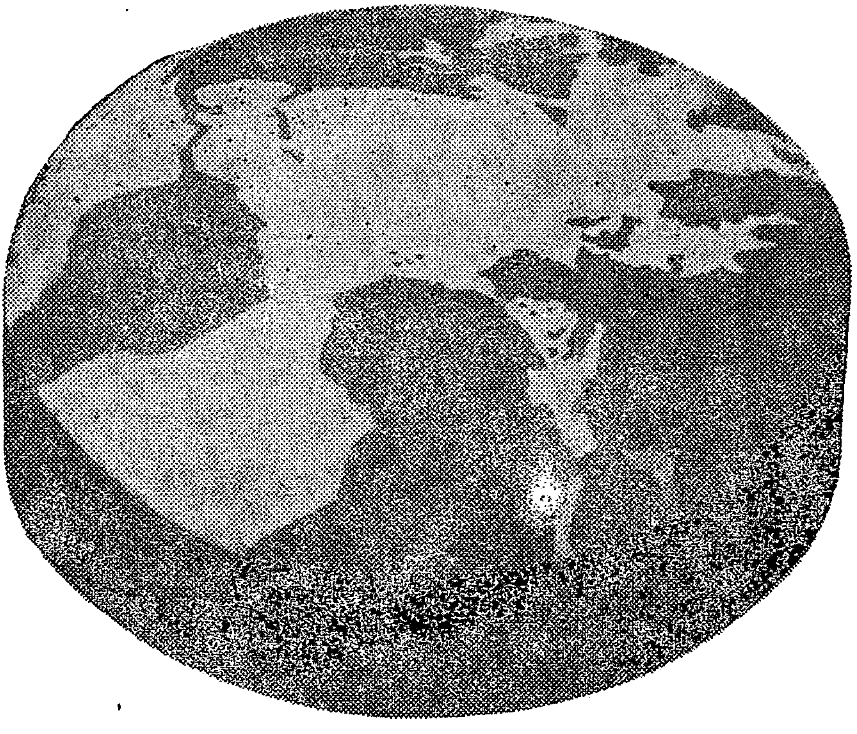

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AMOEBIASIS : SURGICAL ASPECTS

BY

Lt Col G. S. Srivastava, AMC*

INTRODUCTION

Amoebiasis is the most widely distributed of protozoal diseases in the world. In India it is found almost everywhere but in the eastern regions of Bengal, Assam, and NEFA it is endemic and it assumes greater importance in the Armed Forces because a large number of our troops is deployed in these areas.

At least 25% cases of amoebic dysentery as per western literature may develop one of the complications requiring surgical intervention, though no such statistics are available in Indian literature.

CLASSIFICATION

The surgical complications can be classified as under :—

Intestinal

1. Perforation
   - (a) Intraperitoneal
   - (b) Retroperitoneal
2. Haemorrhage
3. Typhilitis/Appendicitis
4. Amoeboma
5. Fistula
   - (a) Coloenteric
   - (b) Colocutaneous

Hepatic

1. Liver Abscess
2. Rupture of Abscess
   - (a) Enteric
   - (b) Peritoneal
   - (c) Pleuropulmonary
   - (d) Pericardial

Rare Complications

1. Amoebiasis Cutis
2. Genitourinary
3. Pulmonary
4. Cerebral

INTESTINAL COMPLICATIONS

1. PERFORATION

A 24 year old soldier while serving in Naga Hills was treated in MI room for three days with thalazole tablets for mild dysenteric symptoms of blood and mucus in stools without any response. He was admitted to hospital where a diagnosis of Ulcerative Colitis was made. Next day he developed sudden abdominal distension with toxaemia and shock. Plain X ray abdomen (Fig. 1) showed gas under the diaphragm. At laparotomy multiple perforations in the caecum and transverse colon were found. As the patient's condition was poor, abdomen was closed with drains. He died within 24 hrs. He did not receive any antiamoebic treatment because amoebiasis was not suspected. This in fact was a case of intraperitoneal perforation of amoebic ulcers in the colon which is a rare but serious complication of amoebic dysentery. Two clinical types of peritonitis due to perforation of amoebic ulcer in the colon are recognised.

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(a) **Intraperitoneal Perforation :**

(i) In the first type, the patient with severe amoebic dysentery develops peritonitis gradually with marked absence of any sudden episode suggestive of acute perforation. Xray abdomen demonstrates gas under the diaphragm but at laparotomy site of actual perforation in the colon may not be found. The treatment, therefore, in such cases should initially be conservative with intensive antiamoebic and antibiotic drugs, nasogastric suction, and IV fluids. In severe cases or when diagnosis is in doubt, laparotomy for peritoneal toilet and drainage is indicated.

(ii) In the second type a case with mild dysenteric symptoms or no symptoms develops a sudden episode of abdominal pain with all the signs of peritonitis and progressive toxicity. Treatment is operative, but time should be spent to resuscitate the patient with IV fluids and antiamoebic antibiotic drugs should be instituted immediately. The perforated segment of bowel should be exteriorised if possible and a proximal ileostomy or colostomy provided to divert the faecal stream, because simple closure of perforation usually gives way and results in a faecal fistula. Occasionally perforations are multiple, involving the entire colon ; in such cases an ileostomy with drainage of the peritoneal cavity in both flanks is probably the only procedure possible.

(b) **Retroperitoneal Perforation :**

These are commoner than intraperitoneal perforations. The usual sites are caecum, ascending colon, and rectosigmoid. Small leaks from retroperitoneal perforation will often seal and heal with antiamoebic and antibiotic treatment; larger perforations result in retrocolic or paracolic abscess which need drainage.

2. HAEMORRHAGE

Massive melœna from a rapidly spreading ulcer eroding a large artery is rare. Treatment should be conservative with repeated blood transfusions because location of the site of bleeding in an edematous colon full of blood is extremely difficult. However, in continuing and life-threatening haemorrhage, emergency total colectomy may prove life-saving.

3. AMOEBIC TYPHILITIS AND APPENDICITIS

Pain and tenderness starting from and confined to the caecum with recent or remote history of diarrhoea or dysentery is characteristic of amoebic typhilitis and should be treated conservatively with antiamoebic drugs, which will produce a dramatic response within 48 to 72 hrs of such treatment.

In the absence of diarrhoea or dysentery this may be confused with appendicitis and surgery undertaken with disastrous results. In case the abdomen is opened on the mistaken diagnosis of acute appendicitis, and the inflammation from the appendix is found to extend onto the caecum, or inflammation from the caecum extends to the appendix, appendicectomy must not be performed, otherwise an inevitable faecal fistula would develop. The abdomen should be closed with a drain at the site and antiamoebic and antibiotic treatment should be instituted. If, however, the inflammation is confined to a small area of the caecal wall around the base of the appendix, or if perforation is threatened, the appendix can be removed along with a cuff of the unhealthy caecal wall around the base of the appendix and the resultant caecotomy closed in two layers and antiamoebic treatment in addition to antibiotics should be given.

4. AMOEBOMA

These are firm granulomatous masses containing multiple abscesses which occasionally develop in the wall of large bowel anywhere from caecum to rectum. Clinically they present as mildly tender masses in the line of the colon in a patient with recent or remote history of dysentery or who has visited an endemic area.

Differential diagnosis lies between Tuberculosis, Crohn's, Diverticulitis, Actinomycosis, and Carcinoma. Ba enema or sigmoidoscopy are not helpful in differentiating one from the other ; even presence of amoebae in the stool does not rule out the possibility of a coexistent carcinoma. Treatment should be a course of antiamoebic drugs for three to six weeks, and the patient should be kept under observation. Doubt in diagnosis at any stage calls for a laparotomy and excision biopsy of the entire mass. Other indications for operation are development of complications like, intestinal obstruction, perforation and fistula formation.

Illustrative Case :—Rarely an amoeboma may present with intestinal obstruction, as it did in this old man of 60 yrs (Fig. 2). Plain Xray abdomen showed distended loops of bowel. At laparotomy a hard mass was found in relation to the caecum and ascending colon. Suspecting a carcinoma, a Rthemicolectomy was carried out. The specimen on opening after operation showed multiple large ulcers in the ascending colon besides the hard mass at the ileocaecal junction. The patient was immediately put on antiamoebic therapy but despite this, he had a burst abdomen on the 7th and a faecal fistula on the 9th post operative day. Burst abdomen was closed and the faecal fistula closed in two weeks under continued antiamoebic therapy.

5. FISTULAE

(a) **Coloenteric :**

These are rare and result from necrosis of deep ulcers or amoebomas lying adjacent to loops of small intestine. Two clinical groups are recognised.

(i) In the first there are symptoms suggestive of partial intestinal obstruction with diarrhoea. Ba enema may show an obstructed segment but fails to demonstrate the fistula unless large. Most of these close with medical treatment, in persistent cases surgery is indicated after a full course of antiamoebic drugs.

(ii) In the second, symptoms are those of localised or generalised peritonitis due to leakage at fistulous site. Patient is toxic and in peripheral circulatory failure. Such a patient should first be resuscitated and managed medically until his condition permits a proximal divertional colostomy under local anaesthesia. Prognosis is bad and mortality high in this group as against the first in which the prognosis is good.

(b) **Colocutaneous** :

Colocutaneous fistulae follow and appendicectomy or colonic resection in an unsuspected case of amoebic colitis or a penetrating injury through caecum or colon in an amoebic patient. Diagnosis is by demonstrating amoebae in scrapings from the fistulae. With amoebicidal drugs the skin lesions clear and the fistula bearing segment of bowel can then be excised and bowel continuity restored.

HEPATIC COMPLICATIONS

1. LIVER ABSCESS

Clinically a painful tender enlarged liver with fever should favour the diagnosis of

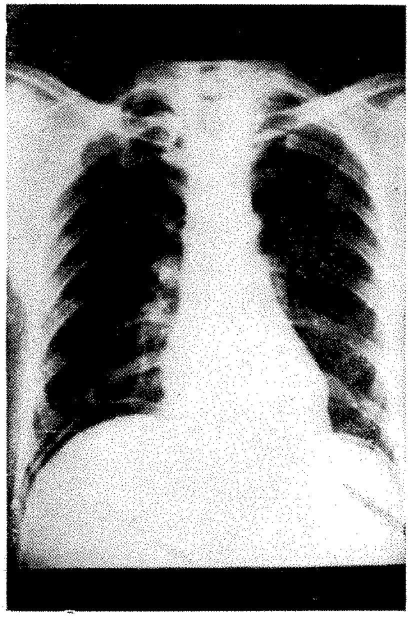

Fig. 1—Showing gas under the diaphragm due to perforated ulcers in the colon.

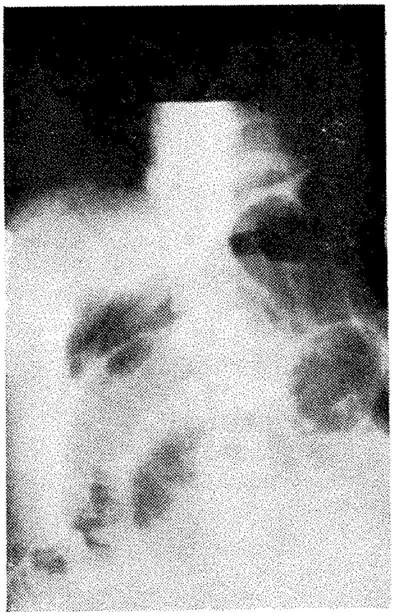

Fig. 2—X-ray abdomen showing distanded loops of bowel due to obstruction at Ileocaecal junction by amoeboma.

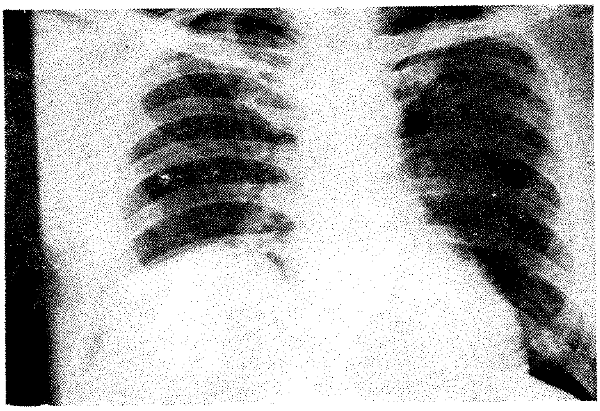

Fig. 3—X-ray Chest showing raised Rt dome of Diaphragm due to liver abscess.

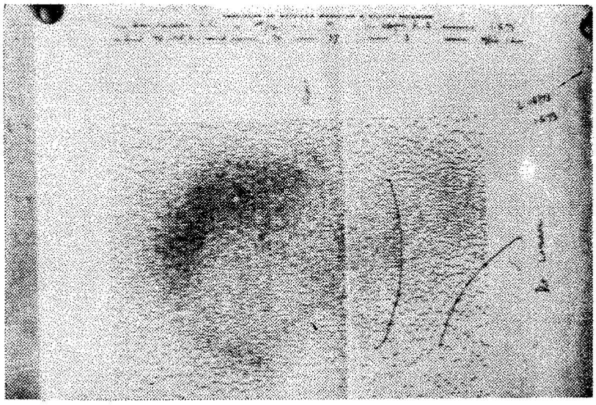

Fig. 4—Hepatic Scan showing Abscess in posterior part of Rt lobe (Lat.)

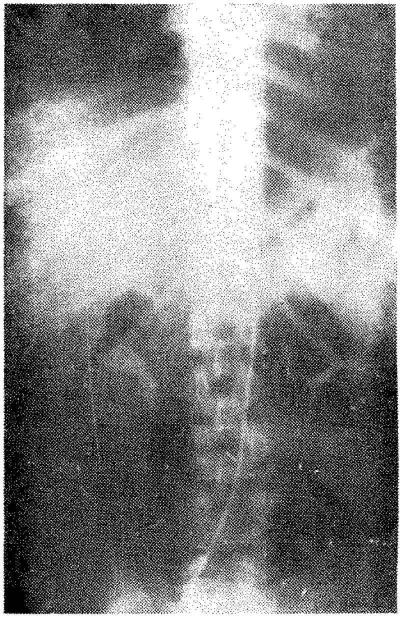

Fig. 5—Coeliac axis angiogram showing displaced branches of Rt Hepatic artery due to abscess.

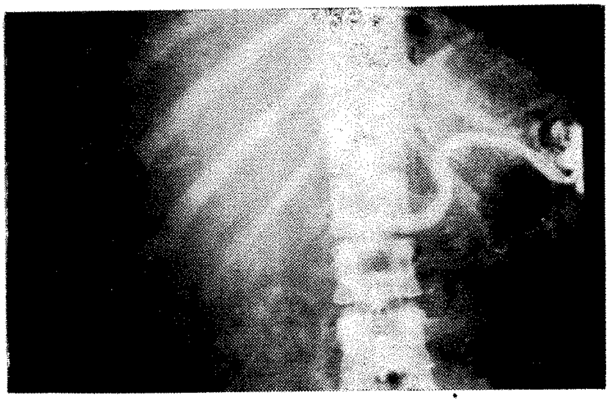

Fig. 6—Splenoportogram showing obliteration of portal veins by abscess in Rt lobe of liver.

amoebic abscess, history of dysentery in the past is usually present. In deep seated abscess the pain and tenderness are not localised. Pain is usually dull, but pressure over the related intercostal spaces elicits extreme tenderness. When diaphragm and pleura are involved in the inflammatory process, pain in the Rt shoulder and cough may be present.

Diagnosis is confirmed by limited movement of the diaphragm on screening and raised Rt dome of the diaphragm (Fig. 3). Dramatic response to Emetine or Metronidazole within 24 to 48 hrs is further evidence of the diagnosis, which is finally clinched by aspiration of 'anchovy sauce' like pus. The pus does not contain amoebae and is sterile unless infected by bacteria. In doubtful or deep seated abscesses other diagnostic aids include Liver Scan (Fig. 4), Selective Coeliac Angiogram (Fig. 5), and Splenoportogram (Fig. 6), which can be carried out at well equipped hospitals. A useful method of demonstrating the size and extent of an abscess cavity is to inject Hypaque into the abscess cavity after aspiration and taking Xray pictures. (Fig. 7).

Epigastric tenderness with fever, weight loss and anaemia should raise the suspicion of a left lobe abscess. If the diagnosis remains in doubt and the abscess cannot be hit by the aspirating needle, exploratory laparotomy and aspiration drainage is mandatory as mortality due to undiagnosed liver abscess is considerably high.

Treatment :—Initially the treatment is medical. Aspiration is indicated when a large swelling in the Rt hypochondrium becomes evident, or there is marked tenderness, bulging, edema, and local signs of inflammation in the intercostal spaces or if significant clinical improvement is not evident within 2 to 4 days of antiamoebic treatment. Closed aspiration is preferable to open drainage but once bacterial infection has occurred open drainage should be provided. Percutaneous needle aspiration of a Lt lobe abscess is contraindicated because of its deep situation and danger of spillage in the peritoneal cavity; it should be aspirated under direct vision with a needle at laparotomy.

2. RUPTURE OF LIVER ABSCESS

(a) **Enteric :** Occasionally a liver abscess may burst into adherent bowel and discharge its contents into duodenum, stomach, ileum, or colon. There is little evidence of a liver abscess, and low grade fever, weight loss and anaemia may simulate some malignant process. Ba meal examination may show an abscess with the liver at its base. Most of these heal with specific chemotherapy; in doubtful cases laparotomy is justified.

(b) **Peritoneal :** Rupture in the peritoneal cavity presents with clinical features no different from those of peritonitis due to perforated peptic ulcer, but a history of dysentery, enlarged liver, and intercostal tenderness may point to the diagnosis. Absence of free gas in the peritoneal cavity in presence of generalised peritonitis on straight Xray of the abdomen helps to distinguish from gastrointestinal perforation. Laparotomy is indicated to confirm the diagnosis and for peritoneal toilet, complete aspiration of the abscess cavity, and drainage of the peritoneal cavity. Intensive antiamoebic and antibiotic therapy is instituted.

Lt lobe or caudate lobe abscess may burst into the lesser sac and present as an epigastric mass; this is typically localised and chronic. This should also be dealt with by a laparotomy and aspiration under direct vision.

(c) **Pleuropulmonary :** Sudden rupture through the diaphragm into the Rt pleural cavity presents with chest pain, fever, toxicity and chest signs clinically and radiologically. The patient may cough out typical 'anchovy sauce' like, or bile stained sputum and this may show amoebae under the microscope. (Fig. 8). Management initially is conservative with antiamoebic drugs but later thoracocentasis may be required to evacuate the pus. Recovery is 85 to 90% with adequate specific chemotherapy.

(d) **Pericardial :** An extremely rare condition. Slow penetration may be indicated by substernal pain, dysponea on exertion, signs of pericarditis, pulsus paradoxus elevated venous pressure, and ECG changes. Screening and Xray are helpful. Treatment is aspiration of the pericardial sac and antiamoebic drugs with care of the left lobe abscess from where the penetration occurs. Sudden rupture is usually fatal unless immediate aspiration of the pericardial sac is undertaken to relieve cardiac tamponade.

RARE COMPLICATIONS

1. AMOEBIASIS CUTIS

This rare condition occurs in the skin surrounding a colocutaneous fistula or on the perineum or genitalia from amoebic lesions of the lower intestinal tract. There is synergistic amoeba-bacterial invasion and sloughing of skin and subcutaneous tissues. The treatment is initially medical and later, scar tissue excision and skin grafting.

2. GENITOURINARY AMOEBIASIS

Extremely rare. Bladder lesions do not develop due to deleterious effect of urine on amoebae. Perinepheric abscess, renal abscess, amoebic nephritis, pyelitis, urethritis, cervicitis and cervical amoeboma have been reported. Treatment is by specific chemotherapy, surgical intervention seldom being required unless rectovaginal or rectourethral fistulae develop, and these should be tackled after eradication of the amoebae.

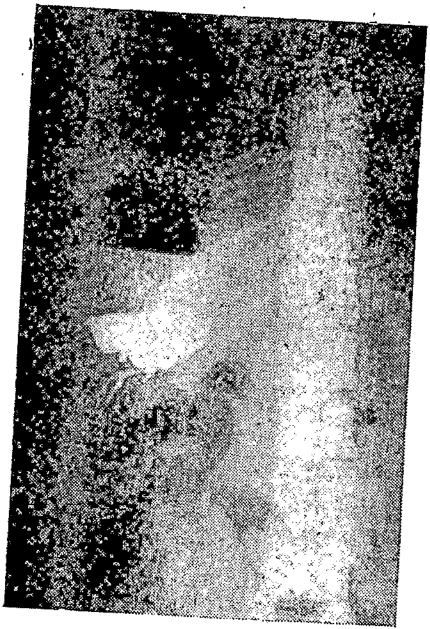

Fig. 7—Abscess Cavity in Rt lobe demonstrated by dye and air injection.

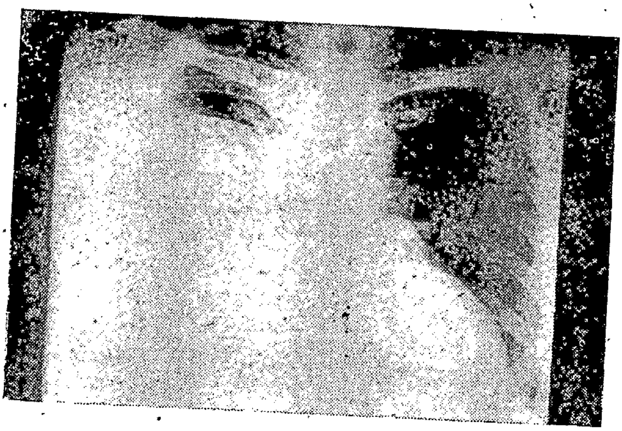

Fig. 8—X-ray chest showing rupture of Rt lobe abscess in Rt. pleural cavity.

3. PULMONARY AMOEBIASIS

Embolisation of amoebae from hepatic veins is responsible for metastatic lung abscesses. There may be haemoptysis, low grade fever, cough, dyspnoea, and chest pain. Xray chest shows an abscess cavity. The abscess may burst into a bronchus resulting into spontaneous cure, otherwise it may resolve with antiamoebic drugs, surgery being rarely required.

4. CEREBRAL AMOEBIASIS

Metastatic Brain Abscess is an extremely rare complication. Just over 100 cases are so far reported. Clinical picture is of a rapidly progressive brain lesion with death within a few weeks. Diagnosis is rarely made before autopsy, and amoebic abscess of the liver or lung is almost always present. In case diagnosis is made or suspected the patient should receive intensive antiamoebic therapy, and aspiration drainage through burr holes provided the abscess can be located. Mortality is extremely high whatever method of treatment is adopted.

SUMMARY AND CONCLUSION

If complications requiring surgery are to be prevented, and mortality rate in those undergoing surgery eventually is to be improved, the following surgical principles must be followed.

1. Intensive and adequate antimoebic therapy must be given to all cases of intestinal and hepatic amoebiasis, as these cases are potential candidates for surgery.
2. Operations on known and suspected cases of amoebiasis should be deferred until 3 to 4 days of antiamoebic therapy has been given.
3. If operation becomes imperative, colostomy or bowel resections should be avoided as these may lead to suture line break-down and result in faecal fistula.
4. If resection is unavoidable, a proximal ileostomy or colostomy should be provided to divert the faecal stream.

ACKNOWLEDGEMENT

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CONCLUDING REMARKS OF THE CHAIRMAN

A high index of suspicion and awareness of the atypical presentation are important to prevent diagnostic and therapeutic pitfalls. Mild or absent pyrexia, insignificant leucocytosis, minimal hepatic enlargement are common examples. It is fortunate, however, that even in such cases there is usually some clue or the other which points to the underlying amoebic nature of the disease. Loss of a case through complications of amoebiasis should be attributed to neglect or to professional incompetence unless proved otherwise.

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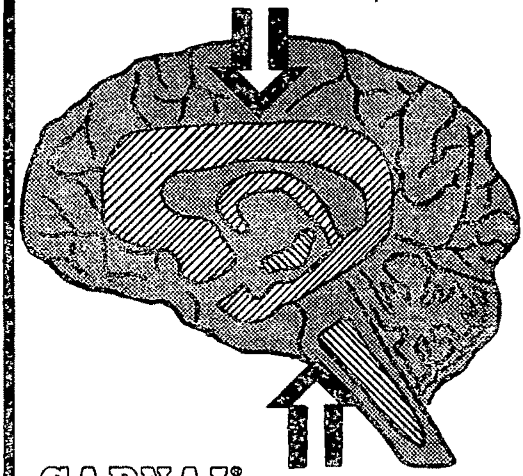

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3 BROTHERS

HAZARDS OF DRUG THERAPY

BY

Brig S. N. Basu*

GENERAL INTRODUCTION

In the 4th century B.C., Hippocrates gave sound advice regarding treatment of a case : "If you can, help; if you cannot, do no harm."

It is difficult to guess what the great physician had in his mind when he said this. It is certain, however, that he knew of the possibility of drugs doing some of the harm to patients seeking medical aid. With better understanding today, it is recognised that drug hazards can be due to toxic reactions, idosyncracy, allergic manifestations, drug interactions and hazards through mutations and genetic effects.

The Science of Medicine is a dynamic one and considerable progress has been made in the fields of pharmacology, therapeutics and allied disciplines. We have learnt a lot about the mode of action, toxicity and side effects of drugs. During the last 20 years or so, knowledge in this field has tended to be more precise. This has been helped by better analytical methods and animal experiments, physiological, biochemical, tissue culture, genetic and enzyme studies.

The spurt in intensive drug study has been attributed to two main factors :

(a) The thalidomide tragedy of 1956–60 when babies were born with congenital defects like phocomelia, to mothers who had the drug during pregnancy.

(b) The introduction of a large number of drugs with consequent temptation of the practice of polypharmacy.

It is desirable to review the advisability of long term therapy with certain drugs, especially the newer ones about which our experience is limited. This is imperative in view of the fact that the concept about action of certain drugs has received a rude shock as a result of controlled studies carried out in many parts of the world.

To illustrate the point, the case of anticoagulant therapy may be considered.

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Some years ago, withholding anticoagulant therapy in a case of ischaemic heart disease was viewed as "just short of criminal negligence". But now the hazards of anticoagulant therapy are better understood and the possibilities of a bleed from peptic ulcer/hiatus hernia, haemarthrosis and cerebral haemorrhage are well appreciated. It is recognised that a large part of the anticoagulants like Warfarin gets bound to the plasma protein. Only the free moiety is pharmacologically active. Drugs like clofibrate (Atromid), indomethacin, salicylates or a sulphonamide when used simultaneously, displace the protein-bound moiety of anticoagulant therapy thereby enhancing its anticoagulant action with the possibility of a serious haemorrhage.

On the other hand many sedatives, tranquillisers and anti-depressants such as barbiturates and meprobate inactivate oral anticoagulants through enzyme induction. Their withdrawal or intermittent use may provoke serious haemorrhages. The exhibition of an anticoagulant is therefore a matter of selection and discretion, not a mere routine. Similarly a doctor should be extremely cautious in prescribing drugs for pregnant women in the first trimester. He should not routinely prescribe sedatives or tranquilliser.

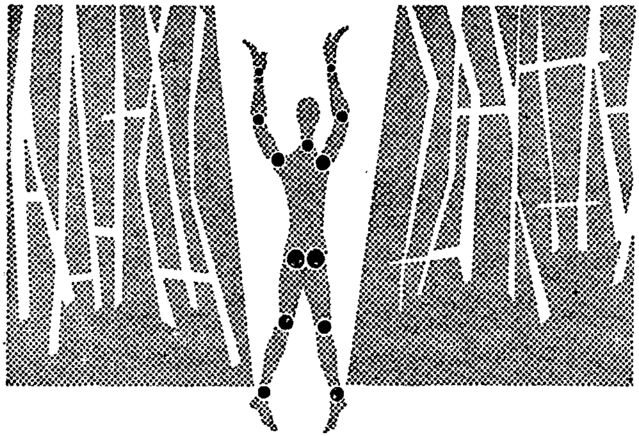
The terratogenicity, hepato, nephro and myelotoxicity of some of the more commonly used drugs is better understood now. The knowledge of enzyme chemistry has helped in the proper understanding of conditions like blackwater fever and other syndromes associated with glucose 6 P. D. deficiency. Hazards of steroids and hypotensive drugs are also better understood now. The practice of poly-pharmacy has brought to light many interesting drug interactions, some of them with dangerous potentialities.

Primary pulmonary hypertension following use of anti-obesity drugs; small bowel stenosis resulting from potassium supplements with long term hydrochlorthiazide; muscle involvement with phenothiazines and interaction of pyridozine and l-dopa are fairly well known today. In spite of these advances a lot remains to be learnt. When Leuenhoek looked through the first working microscope he was overwhelmed with joy on seeing the paramecium and some of the larger bacteria and he shouted in self satisfaction: "I have seen everything that could be seen". Today we know how limited his vision was. In the field of drug therapy some of the safer agents of yesterday are not considered so safe today. The gastroduodenal haemorrhages following aspirin, the nephropathy with phenacetin, nephro-calcinosis with vitamin D, hepatic involvement with paracetamol and thrombophlebitis or liver involvement with "pills" are common knowledge today.

It appears as if no drug is truly safe. The emerging consciousness in this respect should enjoin greater responsibility on the prescribing physician. One must try to know all about each drug he uses. A balanced attitude must be developed and one should always entertain 'new doubts about newer drugs'. Elicitation of the drug history of a patient may be life-saving at times.

It should be the duty of every physician to publicise his experience of a drug hazard so that the others may be alerted.

In conclusion it can be said that use of drugs demands legitimate caution to prevent hazardous side effects.



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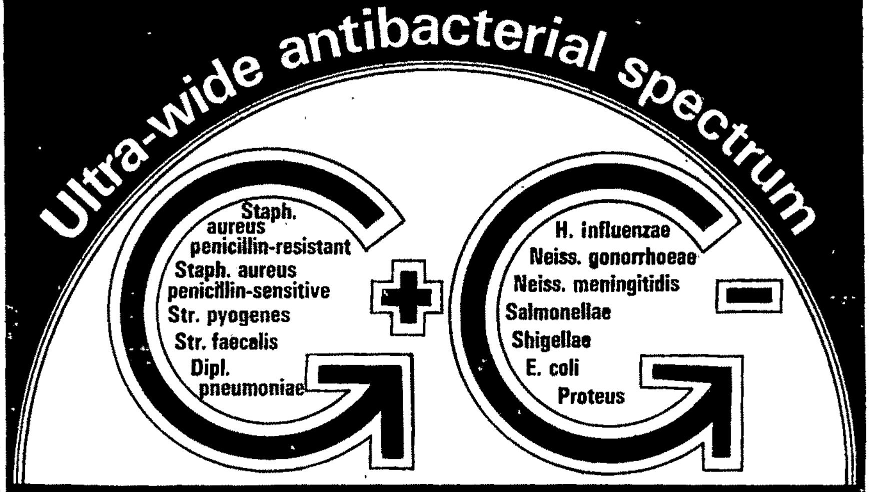

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SENSITIVITY AND TOXICITY OF DRUGS

BY

Colonel K. K. Gupta*

The seriousness of adverse reactions of drugs is seen by the number of deaths on this account in UK, 68 in 1964 and 164 in 1970, (BMJ 1971). Almost any drug may produce unwanted and sometimes unexpected adverse reactions and some of these, such as anaphylactic shock or exfoliative dermatitis, may be hazardous to life. The adverse reactions may be due to hypersensitivity of the body to the drug or due to toxic effect of the drug on the body.

HYPERSENSITIVITY REACTION OF THE DRUGS

The hypersensitivity can be defined as the adverse reactions of the drugs due to an immunological response. The reaction can be immediate or delayed. In immediate reaction (Fig. 1) drug itself or drug protein complex (Hapten) or one of the metabolites of the drug acts as the sensitising antigen. Several antibodies may be formed. Some of them specially, IgE get fixed to various tissues notably to skin and mucous membranes. When the challenging dose of the Antigen (Reagin) is given, it reacts with tissue fixed antibody. This reaction leads to release of SRSA, kinins and histamines causing acute symptoms of broncho-spasm and laryngeal oedema, shock and coma (BMJ 1971). Common drugs that cause immediate reaction are Benzyl penicillin streptomycin and horse serum injections.

In delayed reaction (Fig. 2) antibody (IgM), in the presence of excess of antigen and complement, causes vasculitis leading to erythema of various types, nephritis, arthritis and fever. The delayed reaction can also be due to cell-drug complex acting as an antigen to produce auto-antibodies. These antibodies destroy the antigenic cells. Thus some of the cases of thrombocytopenia, agranulocytosis and haemolytic crisis can be explained due to sensitivity of the drug, (BMJ 1971). Common drugs that cause delayed sensitivity are :—

1. Antibiotics
    - (a) Penicillins.
    - (b) Aminoglycosides, streptomycin, kanamycin, neomycin and gentamycin.

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2. Analgesics	Aspirin.
3. Other Chemicals	Sulpha, butazolidine, thiouracil.

Fig. 1 DRUG SENSITIVITY (Immediate)

Antigens	→ Antibody	→ Release of	→ Clinical
(Sensitising Dose)	(IgE)	Histamine	Urticaria
Drugs	Fixed to tissues	Serotonin	
			Bronchospasm
Drug Protein	Allergin	SRSA	Laryngeal oedema
Complex	(Reagin)		
(Hapten)		Kinin	Tachycardia
Metabolites			Shock
Impurities			Anuria

Fig. 2 SENSITIVITY REACTION (Delayed)

I	II
Antibody (IgM)	Drug-Cell Complex
Antigen Excess	
With Complement	Acts as Antigen
↓	↓
Vasculitis	Auto Antibodies
↓	↓
Urticaria	
Erythema	Thrombocytopenia
nodosum	
Erythema	Haemolytic crisis
multiforme	Agranulocytosis
Nephritis	
Arthritis	
Serum Sickness	
Poly Art. Nod.	
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Clinical manifestations of Drug sensitivity are :—

1. Anaphylactic shock.
2. Asthma.
3. Urticaria and angioneurotic oedema. (Immediate, late or delayed)
4. Non urticarial rashes.

5. Serum sickness syndrome. (Arthralgia, Lymphadenopathy and Fever)
6. Syndromes resembling the collagen disease, e.g., Polyarteritis nodosa; disseminated lupus erythematosus.
7. Severe haematemesis provoked by aspirin.
8. Hypersensitivity effect on individual organs.

Out of these the most dreaded sensitivity reaction of drugs is anaphylactic shock. It occurs most commonly after injection of Benzyl Penicillin or horse serum such as A.T.S. or anti-snake venom, incidence being 1.4 per 1000 and mortality 1.0 per 10,000.

Anaphylactic reaction can begin minutes after administration of the drug and is heralded by feeling of uneasiness, weakness and sweating followed by pruritus and wheal formation, nasal itching and swelling of the face, hands and other soft tissues rapidly. Ominous signs are tachycardia, wheezing, laryngeal stridor, dyspnoea and cyanosis. The patient may have convulsions and pass into coma and shock.

Prevention of anaphylactic reaction due to Penicillin

(a) Limitation of the use of Benzyl penicillin injections to minimum. Penicillin should not be used externally.

(b) Past history of penicillin sensitivity and of allergic diathesis should be asked.

(c) Skin test-0.02 ml of a solution containing 1000 I.U of penicillin G per ml of distilled water, is injected intradermally. Appearance of wheal after 15 minutes is considered a positive test. Incidence of false negative is 14%. Use of PPL (Penicilloyal Polylysine) which is polyfunctional hapten is recommended for skin sensitivity test. Incidence of false negative results with PPL is very low.

Treatment of Anaphylactoid reaction

(a) Patient to be placed in supine position.

(b) Injection of adrenaline 0.2 to 0.4 ml of 1:1000 soln. intramuscularly. If pulse of patient is unobtainable 0.1 to 0.2 ml adrenaline to be given intravenously. Injection can be repeated in 5 to 10 minutes.

(c) Oxygen by mask.

(d) IV administration of 5% glucose.

(e) Diphenhydramine 56 mg IV.

(f) Hydrocortisone succinate 200 to 300 mg IV.

(g) For bronchospasm Aminophylline 500 mg to be added to glucose drip.

(h) If BP remains abnormally low, vasopressor drugs are to be used.

TOXICITY OF DRUGS

Various mechanisms causing drug toxicity on the body are :—

1. Unwanted pharmacological actions.
2. Genetic defects.
3. Drug interactions.
4. Drug tolerance and dependence.
5. Injury to organs—heart, kidney, blood, CNS, Eye & Ears.
6. Foetal damage due to passage of drugs through placenta.
7. Toxic effect on infants due to secretion of drugs through mother's milk.

1. Unwanted pharmacological actions

(a) **Overdosage :** Causes exaggeration of pharmacological actions of drugs. Thus haemorrhage may complicate the use of anti-coagulants or prolonged drowsiness the use of hypnotic drugs.

(b) **Side effects :** Due to unwanted but unavoidable pharmacological action of drugs. Atropine or propantheline used in the treatment of Peptic Ulcer may cause unpleasant dryness of the mouth and blurring of vision. The hypotensive effect of ganglion blocking drugs is inevitably accompanied by undesirable effects due to parasympathetic block. Steroids cause retention of sodium, hypokalaemia, osteoporosis and bleeding from pre-existing peptic ulcer. Chlorthiazide diuretics cause hypokalaemia and diabetogenic tendency.

(c) **Secondary effects :** Secondary effects are the indirect consequences of primary drug action. Examples are, vitamin deficiency or super infection by staphylococcus which may occur in patients whose normal bowel flora has been altered by the antibiotics. The Harxheimer reaction due to products released by killed spirochaetes is another type of secondary effect.

2. Genetic Defects

Haemolytic reaction due to 8-aminoquinoline drugs (primaquine, Pamaquine) is due to deficiency of glucose 6-Phosphate dehydrogenase in red cells. The action of muscle relaxant suxamethonium is prolonged due to abnormality of serum pseudocholinesterase which normally inactivates the drugs. Porphyria after use of sulpha drugs is due to excess of ALA-synthetase.

3. Drug Interaction

Patients receiving multiple drugs can have propensity for adverse reaction. The adverse reaction may be due to ;—

(a) **Interference with absorption :**—For example if calcium antacids and tetracycline are given together, tetracycline absorption will be reduced. Dilantin sodium and nitrofurantoin by inhibiting intestinal conjugates, retard absorption of folic acid and so may cause megaloblastic anaemia.

(b) **By competition of drugs for plasma protein :**—If a patient getting warfarin sodium is given phenyl butazone, the latter drug will combine with plasma albumin and thus induce release of warfarin sodium from plasma albumin and this will precipitate bleeding, (BMJ, 1971). Methotrexate is bound to plasma albumin and can be displaced by various acidic drugs such as sulpha and salicylates thereby enhancing the toxicity of methotrexate.

(c) **By interfering with metabolism of other drugs :**—

(i) Phenobarbitone by its quality of enzyme induction increases the metabolism of warfarin sodium. So in a patient, whose dose of warfarin is stabilised when he is also taking phenobarbitone, the dose of warfarin will be excessive when the latter is stopped and it may cause bleeding.

(ii) Chloramphenicol can inhibit metabolism of Tolbutamide and may thus cause hypoglycaemia. Similarly it inhibits metabolism of hydantoin sodium thus causing nystagmus and other toxic symptoms of anticonvulsants. Monoamine oxidase inhibitors (drugs like Nardil) inhibit metabolism of catecholamines and so sympathomimetic drugs if given alongwith the MAO inhibitors, may cause hypertensive crisis leading to subarachnoid haemorrhage. Diuretics increase excretion of potassium and thus interfere in degradation of digoxin. The hypokalaemia caused by diuretics may cause digitalis intoxication.

4. Drug Tolerance and Dependence

(a) Drug tolerance is defined as need to increase the dose of drug to sustain the initial effect. Tolerance may be either due to (a) enhanced detoxication or (b) cellular adaptation of the neurons to continued high drug concentration.

(b) Drug dependence (addiction) has been defined by W.H.O.(1969) as hazardous effects of the drugs threatening physical health, psychological functioning and adaptation to the demands of the society. Repeated administration of a drug creates an altered physiologic state in the body that requires continuance of the drug to prevent a characteristic physical illness called "withdrawl syndrome".

(c) Drugs producing tolerance and dependence are generally those acting on central nervous system. Common drugs are Opiates, alcohol, barbiturates, chloral hydras paraldehyde, chlordiazepoxide and diazepam amphetamine group of drugs and meprobomate.

Drugs such as Cocaine, hallucinogens-LSD, Tryptamines and Cannabis indica (hashish) are taken for psychic effects and may cause craving but on withdrawal cause no physical symptoms.

5. **Toxic effects on individual organs**

(a) **Liver :**—Sheila Sherlock (1968) described the following hepatic damage as toxic effect of drugs:—

(i) **Direct cell injury:**—Carbon tetrachloride, tetracyclines, DDT, antimitotic drugs and chloroform. Intravenous tetracycline is hepatotoxic, more so in pregnancy. In direct cell injury, there is fatty infilteration and cell necrosis. Clinically there is jaundice with liver enlargement and raised S.G.P.T. Cases may end fatally due to hepatic failure with or without renal failure.

(ii) **Hepatitis like picture:**—M.A.O. inhibitors, isoniazide, pyrazinamide, ethionamide, hallothane and cincophen. Clinical picture is like that of viral hepatitis but mortality is 20% compared to 1% of infectious hepatitis. It is thought that the condition is due to activation of a virus.

(iii) **Cholestatic injury:**—Phenothiazines, steroids, diabinese and P.A.S. Clinical picture is of obstructive jaundice. The block is biochemical, occurring after bilirubin has been conjugated rather than mechanical.

(iv) **Generalised hypersensitivity reaction involving liver:**—Phenothiazine, erythromycin and oleondomycin. The jaundice will be associated with fever, eosinophilia, rash and blood dyscrasias.

(b) **Kidney**

(i) Many drugs or their breakdown products are excreted through the kidney. Their high concentration may damage the renal tubules. Commonest nephropathy is due to analgesics such as phenacetin, salycilates and aspirin. Paracetamol is considered relatively safe. The symptoms of analgesic nephropathy are polyuria, polydypsia haematuria and sterile pyuria. Nacrosis of papillae may cause acute renal failure (Laurence 1971).

(ii) Pre-existing renal damage may cause accumulation of toxic drugs such as streptomycin, neomycin, kanamycine, colistin and gentamycin. If these drugs have to be given in presence of renal damage, reduction in their dosage is effected depending upon the degree of renal failure (B.M.J. 1971).

(c) **Blood**

Blood dyscrasias may be due to direct action on blood cells and bone marrow or haematological reactions may be due to hypersensitivity.

(i) **Thrombocytopenia** :—Sedormid, phenylbutazone, gold, sulpha drugs, antimitotic drugs, troxidone and thiouracil derivatives. In most of the cases drug combines with the platelets and the combination acts as an antigen causing production of antibodies against platelets.

(ii) **Agranulocytosis** :—Chloramphenicol, sulpha drugs, diuretics, anticonvulsants, methyldopa, gold and amidopyrine.

(iii) **Aplastic anaemia** :—Chloramphenicol, sulpha drugs, phenylbutazone, hydantoin, troxidone, gold and DDT.

(iv) **Haemolytic reactions** :—May be due to direct action on RBC as in case of lead and phenylhydrazine. As explained earlier antimalarials may cause haemolysis due to genetic defect such as deficiency of glucose 6-phosphate dehydrogenase. At time drugs and RBC combine and the combination is antigenic. This occurs with phenacetin, PAS, quinine, quinidine and organic antimony compounds.

(d) **Skin**

The dermatological reactions of the drugs are :—

Contact dermatitis :—Local application of antimicrobials or antihistamines.

Acne :—Bromides-iodides.

Fixed eruptions :—Phenolphthalein, sulpha drugs and barbiturates.

Photo allergy :—Sulpha drugs and antimalarials.

Exfoliative dermatitis :—Heavy metals, sulpha and phenylbutazone.

Stevens Johnson syndrome :—Sulpha and barbiturates.

Urticaria and Angioneurotic oedema :—Penicillin, horse serum and aspirin.

Pigmentation :—Progesterone, phenothiazines, chloroquine and arsenic.

Lupus erythematosus :—Hydralazine, sulpha drugs and procainamide.

Alopecia :—Antimitotic drugs and heparin.

(e) **Nervous system**

(i) Commonly used drugs affecting CNS are sedatives and tranquillizers. All of them produce tolerance, dependence psychic or physical. Besides, most of these drugs can cause dementic changes after prolonged use. Heavy metals and vaccination at times may cause toxic encephalopathy.

(ii) Phenothiazines, mercury and cocaine affect extra pyramidal system and produce parkinsonism like syndrome.

(iii) Polyneuropathy can be caused by Isoniazid, nitrofurantoin, streptomycin, sulpha drugs, chloroquine, antimitotic drugs and heavy metals.

(f) **Ears**

(i) Streptomycin acts on VIII nerve and damages vestibular division causing vertigo and deafness. The damage is reversible if the drug is stopped in time. Dihydro-streptomycin damages the cochlear division and irreversible deafness is caused.

(ii) Other antimicrobial drugs causing cochlear and vestibular damage are kanamycin and neomycin.

(iii) Among the antipyretics, quinine and salicylates are ototoxic and give rise to tinnitus which is relieved on withdrawal of the drugs.

(g) **Eyes**

Toxic effects of drugs on Eyes are as follows :—

Keratopathy – Chloroquine.

Retinopathy – Chloroquine.

Optic atrophy – Quinine, Chloroquine, tobacco and methyl-alcohol.

Glaucoma	– Steroids.

6. Toxic effects on foetus

(a) **First trimester** :—Drugs in first trimester cross the placenta and affect the foetus causing teratogenesis. Thalidomide, vitamin 'A' and 5 HT are the known examples. The teratogenic effect of any drug is difficult to predict by animal experiments. So best possible way is to restrict drug therapy to minimum to women of child bearing age (Nelson and Forfor 1971). Morning sickness occurs at the time when foetus is vulnerable, so it is very important to restrict drug therapy to minimum for this symptom.

(b) **Second and third trimester** :—Following drugs can cross the placenta and may affect the foetus adversely :—

Hormones	:—Androgens and progesterone cause masculinisation of female foetus.
Idodide and antithyroid drugs.	:—Foetal goitre.
Tetracyclines	:—Interferc with teeth and bone development.
Antidiabetic drugs	:—Foetal hypoglycaemia.
Streptomycin	:—Damages foetal VIII Nerve.

7. Toxic effects on infant

Following drugs are secreted in mother's milk and cause harm to the infants :—

Thiouracil, radioactive iodine, barbiturates, alcohol, bromides, iodides and penicillin.

CONCLUSION

While using the drugs the doctor's objective should be to obtain maximum good with the minimum harm. All the adverse side effects may not be avoidable but most of them can be prevented by more thoughtful use of drugs. It is essential that pharmacological action of the drugs should be well known to the treating physician so that he can use them only when needed, in minimum effective dosage and can stop them as soon as early toxic symptoms appear.

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DRUG RESISTANCE

BY

Colonel D. S. Rastogi, VSM, Senior Adviser (Chest Diseases)

Many drugs give reproducible responses in the same individual over a prolonged period of time. In other instances, there is a progressive diminution of response to such an extent that the drug may have no action at all. This is known as drug resistance and is a major hazard and tragedy for the patient, since further treatment may be prolonged, expensive and the outcome uncertain.

The examples of Drug Resistance are:—

(a) Isoprenaline and adrenaline in asthma.

(b) Insulin in Diabetes mellitus.

(c) Chloroquin etc in Malaria.

(d) Antibiotics in infections.

Resistance to isoprenaline has been observed in asthmatic patients who have been taking it in high doses for a long time and they show no improvement till the drug is withdrawn. Temporary resistance to adrenaline is commonly seen during status asthmaticus when the patient is severely acidotic.

Insulin resistance is said to occur when more than 200 units of insulin are required daily for more than 48 hrs in the absence of ketoacidosis, infection and other endocrine disorders.

Drug resistant cases of malaria were seen in Viet Nam in fairly large numbers and recently such cases have been found in Eastern Sector of our country.

This paper deals with Bacterial Resistance to Drugs with special reference to tuberculosis.

BIOLOGICAL MECHANISM OF DRUG RESISTANCE

There are several mechanism by which bacteria acquire resistance against antibiotics :—

(a) Spontaneous Random Genetic Mutation.

(b) Adaptive Mechanism of Microbial Enzyme.

(c) Resistance Transfer Factors.

(d) Microbial Persistence.

(e) Cross Resistance.

(f) Natural Resistance.

SPONTANEOUS RANDOM GENETIC MUTATION

The progeny of a normally sensitive organism does not remain homogeneous but becomes a mixed population with some descendents which are not identical with their parents. Due to mutation phenomenon some organisms may develop drug resistance. In normal circumstances these mutants are at a biological disadvantage and cannot flourish but the presence of an antibiotic to which the majority of the colony is sensitive radically changes the state of affairs. As the sensitive organisms are killed off, so the drug resistant mutants grow more and more rapidly until they replace the entire bacterial population and a resistant strain has been achieved.

This can be illustrated by an example of a tuberculous cavity which may contain a mixed population of 10^8 bacilli:—

(i) Largest fraction consists of bacilli sensitive to SM and INH and will be killed by both drugs.

(ii) 1000 Bacilli may be resistant to INH.

(iii) 100 Bacilli may be resistant to S.M.

(iv) Almost none resistant to both INH and S.M. If such a patient is treated by only one drug i.e. INH the sensitive organism will be killed but the resistant 1000 organisms will grow more and more rapidly till they replace the entire bacterial population.

ADAPTIVE MECHANISM OF MICROBIAL ENZYME

Bacteria may produce an enzyme which destroys or neutralises the antibiotic concerned, when they may be called drug destroying bacteria. The best example of this is the production of penicillinase by certain strains of staphylococci. Another example is that of highly resistant tubercle bacilli to INH which are unable to form catalase.

RESISTANCE TRANSFER FACTORS

Resistance transfer factors are present in the cytoplasm of bacteria and are capable of transfering bacterial resistance through cell to cell contact within a few minutes. Drug resistance may be transferred from one bacterial species to another. Recently several cases of chloramphenicol resistant typhoid fever were reported from Kerala where the patients had earlier been treated for shigella and E. coli infections by chloramphenicol.

MICROBIAL PERSISTENCE

This type of drug resistance is due to failure of multiplication with the consequence that the organisms are "physiologically insusceptible" to the action of drugs, which are inhibitory only to multiplying organisms. Microbial persistence is seen in infections occurring in relatively avascular situations such as heart valves and within closed tuberculous caseous lesions. This is the most common mechanism of relapse in subacute bacterial endocarditis and pulmonary tuberculosis.

CROSS RESISTANCE

Cross resistance between two antibiotics means that micro organisms that have resistance to one will automatically be resistant to the other. It may be complete or partial and is usually found between drugs with a similar structure i.e. penicillins, sulphonamides and tetracyclines. Partial cross resistance exists between ethionamide and thiacetazone and between kanamycin and viomycin.

NATURAL RESISTANCE

Bovine strains of tubercle bacilli are found to be naturally resistant to PAS and pyrazinamide.

ACQUIRED DRUG RESISTANCE

The incidence of acquired drug resistance to tuberculosis is increasing in developing countries but diminishing in technically advanced countries. Reports from various centres in India suggest that at least 50% of patients first seen in hospitals with a previous history of chemotherapy are excreting resistant organisms. This is an alarmingly high incidence.

CAUSES OF ACQUIRED DRUG RESISTANCE IN TUBERCULOSIS

(i) Failure to prescribe the optimal chemotherapy. Lack of money/medical facilities.

(ii) Failure on the part of patient to take the drugs as prescribed.

(a) Psychological factors which cause many patients to revolt against prolonged self medication.

(b) Failure to appreciate the need to continue taking drugs which may be unpleasant long after they have lost all symptoms of disease.

(c) Unpleasant side effects.

(d) Forgetfulness and failure to understand instructions.

PRIMARY DRUG RESISTANCE

This implies that the organism is resistant to an antibiotic with which it has never come into contact. It may result in failure to respond to the drug and may lead to a further resistance. In case of tuberculosis, a high or increasing prevalence of primary drug resistance suggests the spread of drug resistant strains from unsuccessfully treated patients and is a warning of the inadequacy of treatment.

DIAGNOSIS OF DRUG RESISTANCE IN TUBERCULOSIS

(a) Evidence before treatment:—

Accurate history of previous chemotherapy. Greater the period for which unsuccessful chemotherapy has been administered the greater the probability that drug resistance has emerged.

(b) Evidence during chemotherapy :—

(i) Clinical examination.

(ii) Serial X-Ray show progression of lesion.

(iii) Monthly quantitative sputum smear show increase in the number of bacilli.

(iv) Laboratory sensitivity test is confirmatory but takes three months.

Prevention of drug resistance in Tuberculosis

The following measures are effective against this socio-economic problem.

(a) Motivation and education.

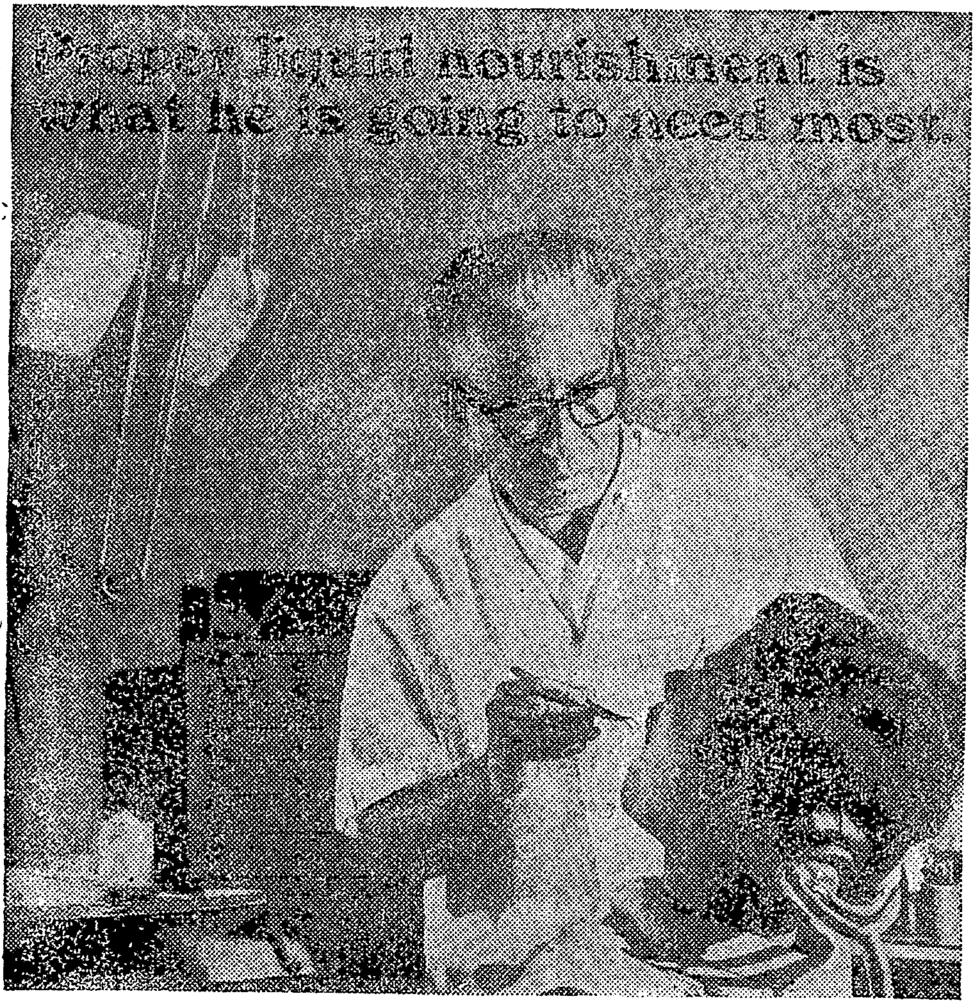
(b) Correct regime of drugs. To be always taken in combination and never singly.

(c) Correct dosage of drugs.

(d) Intermittent therapy should be fully supervised.

(e) Adequate duration. Minimum duration of treatment is 18 months.

(f) Consider surgery in selected cases. A solid residual lesion will not clear with drugs alone and may later be a cause of relapse.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THE ROLE OF A NURSE IN THE RECOGNITION AND MANAGEMENT OF DRUG REACTION

BY

Lieut (Miss) P.N. Rajeswari, B.Sc. Ng.*

INTRODUCTION

The role of a nurse in the recognition and management of drug reactions is a very important one. The nurse by virtue of the nature of her duties, involving long hours in the ward and administration of drugs, has the advantage of being closer to the patient than the doctor. Thus she is the one who sees more of the patient and is the most likely person to notice any change in him. Moreover with her sympathy and better rapport, the patient is more likely to tell her of any particular symptom he may have developed with a drug. Since certain drug hazards can lead to fatality very rapidly, it is absolutely essential for every nurse to anticipate and recognise possible hazards and to institute immediate and effective treatment, to preserve life before the doctor arrives.

The nurse must know the method of administration, dosage, therapeutic effects, side effects and the toxicity of the drugs used in patients under her care. On no account should the nurse take on the responsibility of the doctor to administer drugs on her own. Even apparently safe drugs can produce dangerous effects under certain circumstances. For example a patient on anticoagulants may complain of a headache and the kindly nurse with all good intention may give him an analgesic like aspirin which can be dangerous in such cases as this can induce a haemorrhage. Alternatively headache in a patient on anticoagulant therapy may be the first symptom of a haemorrhage in the brain. Addition of aspirin may further enhance the haemorrhage.

Accuracy has to be stressed. The nurse should be absolutely sure of the right medication and the right amount being given to the right patient. Patients are not necessarily responsible people and the drugs should not be kept with them. It is the duty of the nurse to administer the drugs personally and remain with the patient until the drug is taken. She should record all medication after they are given. No nurse should give a drug prepared by someone else and she should not chart a medication as given unless she has personally given it.

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* Comd Hosp (SC) Pune

If the nurse has any doubt as to the dosage, she should not hesitate to consult the doctor. A mistake may mean death. It is better not to give the drug at all than to give the wrong one or in the wrong amount.

Recognition and management of Drug Haz ards

Recognition and management of drug hazards are possible only if the nurse makes a keen observation on her patients getting drugs which are known to have side effect. It will not be possible to cover all the drug hazards in this short paper. Only a few important examples will be cited.

Antibiotics

They are used very commonly in our hospitals. They produce in some patients an allergic reaction which varies from a burning sensation at the point of contact to a severe dermatitis and even anaphylactic shock and death.

Anaphylactic Reaction

One of the most important and dramatic reactions both in onset and termination, is anaphylactic reaction. Much can be done by the nurse who is at the patient's bed side and by whose administration the events may have followed.

(a) Prevention :—Avoid using drug to patients with a history of hay fever, asthma or other allergic disorders unless necessary. Whenever possible enquire if the patient has had the drug previously. Whenever there is a history of allergic reaction on previous occasion, do not give the drug either orally or by injection. We usually carry out an intradernal skin test before giving penicillin or other serum injections but this test is not infallible. The patient may not show any sign of allergy on sensitivity test but may develop one after three or four injections. Hence the past history of previous injections or reactions may become important.

(b) Recognition :—The patient may first complain of itching at the site of injection which becomes generalised. There is extreme restlessness and marked dyspnoea may appear. Severe shock and loss of consciousness may follow so soon that one should always be prepared for the emergency.

(c) Management :—Every ward must keep an anaphylactic tray and it is imperative that the nurse on duty knows the use of each item in the tray. She should check daily that her emergency tray is complete with all essential drug and equipments.

(i) Injection adrenaline 1 : 1000 is the sheet anchor of treatment. 1 ml I M is given at first. If the patient does not respond immediately then dilute 1 ml of 1 : 1000 adrenaline in 10 ml of saline and give this I.V. very slowly. Minimum 5 minutes should be taken.

(ii) If the response is inadequate hydrocortisone hemisuccinate 100 mg in IV drip may be given.

(iii) Place the patient in shock position raise the foot end of the ebd.

(iv) Maintain adequate airway.

(v) If bronchospasm is severe I V aminophyllin is given very slowly as it can also lower B.P. Oxygen may be administered as required.

Gastric Irritants

Another very common drug hazard is irritation of stomach and haematemesis by common drugs like aspirin, butazolidine, indocid and corticosteroids which are used in abundance in our hospitals. One should never administer these drugs on an empty stomach and one should always anticipate bleeding in the G I tract if these drugs have been given in large doses or for a long time. Slight pallor and a rising pulse should arouse suspicion. The stool should be examined microscopically. These can give information in the form of dark tarry stools. Stool should also be sent, once or twice, for occult blood test.

Untoward Effects on Bone Marrow

All patients getting butazolidine, sulphonamides, antithyroid drugs, chloramphenicol etc should be watched for agranulocytosis. Weekly blood count may not help as sometimes symptoms may appear suddenly. One should watch for sorethroat, fever and lymph glands in the neck. The offending drug should be stopped at the first suspicion.

Sulphonamides

Patients getting sulphonamides should be watched for untoward symptoms, the commonest being drug fever and skin rash. Haematuria can occur leading to oliguria and anuria. This is due to the drug getting precipitated mainly in the collecting tubules and calyces.

Ensuring an adequate urinary output is very important. Instruct the patient to consume plenty of fluids. It is the duty of the nurse to see that he consumes adequate amount of fluids. Ideally a record should be kept of his fluid intake and output.

Timing of drug Administration

Timing of administration of drugs is important. For example ephedrine should not be given late in the evening as it can cause insomnia. Similarly routine diuretics should preferably be given in the morning to avoid disturbing the patient during the night. Patients getting diurectics should be replenished with potassium, as they lose potassium excessively. This is especially important when the patients are on digitalis as hypokalemia leads to digitalis toxicity. Early toxic effects are loss of appetite, nausea, vomiting, abdominal discomfort and occasional diarrhoea. One should watch the pulse and note any unusual bradycardia or irregularity. If these abnormalities appear next dose of drugs should be omitted and the physician informed.

Anticoagulants

Mention has already been made about anticoagulants. One should anticipate bleeding and any abnormal bleeding should be reported to the physician immediately. This includes rectal bleeding, vaginal bleeding, prolonged menstrual flow etc. Gums and skin should also be examined for evidence of bleeding. Gentleness should be ensured in giving mouth care. Skin too needs to be watched especially the injection sites. As the dose of anticoagulants is adjusted by prothrombin time, one should ensure that the correct amount of blood is drawn at the correct time and sent to the laboratory. If the quantity of blood is not correct prothrombin time result will be faulty leading to wrong dosage of anticoagulant. If the prothrombin time is double the control next dose of anticoagulants should not be given and the medical officer should be informed. Next sample of urine should be sent for microscopic examination.

Intravenous therapy

Parenteral fluids have to be given rapidly in shock due to haemorrhage and dehydration. But when the blood volume is not diminished a rapid transfusion may be fatal due to pulmonary oedema, which is recognised by increasing breathlessness, cough, frothy expectoration, rapid pulse and cyanosis. In severe anaemia transfusion should be slow and preferably of packed cells. Danger may further be reduced by giving a rapid acting diuretic like frusemide. The particular label on the bottle like the name of the patient, blood group etc should be read thrice to avoid error in transfusion.

Terratogenic effect of the drugs

To prevent terratogenic effect no drugs should be given to mothers in the first trimester unless specially prescribed by the doctor. Since morning sickness of pregnancy occurs during the time when the foetus is most vulnerable, it is especially important to restrict drug therapy of the symptom to a minimum.

Conclusion

The treatment of a case in the hospital is a team work and the nurse plays an important role in the team. She is the vital link between the patient and the doctor. Osler once said "Listen to the patient, he is telling you the diagnosis" and very often the diagnosis is first made by the nurse. Another responsibility of the nurse, as said earlier, is the correct administration of drugs.

"If the Nurse gives the right dose of the right medicine to the right patient at the right time in the right manner an error in medication is impossible". This quotation is the one that every Nurse should learn and remember.

RECOMMENDED READING

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3. R. S. Satoskar : Pharmcology and Pharmacotherapeutics, Popular Prakashan, Bombay, Ed 1973, Pages 291-422
4. Pearce Evelyn : A General Text Book of Nursing, Faber and Faber Ltd, Lucknow, Ed 1963, Pages 231-253

CONCLUDING REMARKS OF THE CHAIRMAN

Drugs are a double-edged weapon. Carefully used they may be life saving. Indiscriminately administered they may pose serious hazards not only to the patient but also to the offsprings. Toxicity, allergy, hypresensitivity and tolerance have been widely recognised. Drug interaction merits deeper thought and consideration. The pernicious practice of polypharmacy needs to be curtailed.

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PANEL DISCUSSION ON RENAL FAILURE

Chairman : **Brig R Ganguly, AVSM, VSM, Professor of Surgery, AFMC, PUNE**

Members : Gp Capt S Krishnamurthy, Assistant Professor, Department of Medicine, AFMC, PUNE

Colonel Y Singh, Assistant Professor, Department of Surgery, AFMC, PUNE

Lt Col SD Khanna, Professor, Department of Obst & Gyn, AFMC, PUNE

Lt Col NN Sharma, AVSM, Classified Specialist (Medicine), Army Hospital, Delhi Cantt

Lt Col J. Vasudevan, Reader, Department of Medicine, AFMC, PUNE

Major M. Kumar, Classified Specialist (Surgery), Army Hospital, Delhi Cantt.

CHAIRMAN : Ladies & Gentlemen,

Our panel discussion today on Renal Failure is of vital interest to every clinician, especially for those who have encountered gravely ill patients, whose kidneys have failed to function. The subject is not only complicated but also most controversial. The members of the panel are very experienced clinicians, since 'Renal Failure' is a clinical problem.

The primary function of the kidneys is the removal of metabolic waste of body fluids. Renal failure means a failure of this function of the kidneys—either as an acute or abrupt development, or as a slowly progressive chronic affair. But it is not always the kidneys which are primarily to be blamed, and the initial fault may lie outside the kidneys. This is why we have the time honoured etiological classification of pre-renal, renal and post-renal failures. Gp Capt Krishnamurthy, is there any point in sticking to this time honoured classification ?

GP CAPT KRISHNAMURTHY : When renal failure is due to structural damage of kidneys, it is in a sense irremediable, but when the failure is due to extrinsic pre or post-renal cause, appropriate treatment may restore adequate renal function.

CHAIRMAN : I hope you will agree that if you allow any pre-renal cause like hypovolaemia to go untreated, it will surely lead to structural damage of the kidneys. In the same way what is post-renal today will turn out to be renal tomorrow, because any long standing obstruction to urine outflow is bound to produce some renal damage.

GP CAPT KRISHNAMURTHY : One cannot always draw clear cut boundaries and there is bound to be a lot of overlapping. The kidneys have to suffer ultimately, whatever may be the basic initial cause.

CHAIRMAN: In most cases of acute renal failure, the causes are usually pre-renal, which subsequently become renal whereas in chronic renal failure, there is either a basic renal pathology or a definite post-renal obstructive cause.

COL Y. SINGH : In acute renal failure the cause is either renal circulatory insufficiency caused by severe dehydration, haemorrhage and shock, or acute tubular necrosis caused by prolonged renal ischaemia. However, in chronic renal failure, there is either a long standing partial obstruction to renal outflow or a chronic disease of the kidneys with bilateral renal atrophy or fibrosis.

CHAIRMAN : There is no doubt that most of the acute renal failure cases are due to undetected hypotension but severe crushing injuries or tourniquets left too long may also lead to renal shut down. There is such a case on record in an Orthopaedic Centre following three hours of surgery under a tourniquet. We have this problem of acute renal failure in pregnancy and Lt Col Khanna may remember a recent case, where there was a renal shut down after delivery.

LT COL KHANNA : In that particular case the problem was not only that she had one functioning kidney, but even this was the seat of chronic infection. Pregnancy throws an extra load on the kidney. Renal failure may occur in early pregnancy due to septic abortion with severe blood loss, or later on, due to concealed accidental haemorrhage or eclampsia. Renal shut down may also result from severe post-partum haemorrhage not promptly treated.

CHAIRMAN : It is generally agreed that the severe hypotension left untreated may lead to structural damage in the form of acute tubular necrosis. This poses a practical problem of distinguishing between functional oliguria and oliguria due to established renal failure. Lt Col Vasudevan would let us know if the usual test of renal concentrating ability, such as the specific gravity test, is enough to distinguish functional failure from acute tubular necrosis.

LT COL VASUDEVAN : A sure way is to measure the osmolarity of plasma to urine. In physiological oliguria the ratio is usually more than 2, whereas in established failure it is less than 2, but estimation of Na content of urine in relation to specific gravity, if available, may be of great help.

CHAIRMAN : Apart from the routine laboratory reports, I think the overall clinical picture of the patient and his response to treatment may be of greater help. Since long standing obstruction of urine outflow may lead to chronic renal failure, would Major Kumar like to tell some of the important causes where timely surgical intervention may benefit the patient ?

MAJOR KUMAR : As far as the urologists are concerned, the common cause of obstruction of urine outflow leading to chronic renal failure are benign prostatic enlargement or bilateral urinary calculi. Unfortunately, some of these patients remain comparatively asymptomatic and by the time they are diagnosed, they already have some damaged kidney function. In such cases, surgical relief of obstruction is likely to restore kidney function—though in clinically uraemic cases the operation may have to be carried out by preliminary drainage procedures. Other conditions are congenital urethral valves and vesico-uretic reflux.

CHAIRMAN : Lt Col Sharma, what are the usual intrinsic renal causes of chronic failure, where the prospects are not so good and the patient remains a chronic invalid ?

LT COL SHARMA : I think chronic pyelonephritis is the most common cause of chronic renal failure in adults. In the younger age group, we have to think of chronic glomerulonephritis. Less common causes are polycystic disease of kidneys, accelerated hypertension and nephro-calcinosis. The prognosis is not good in such cases.

CHAIRMAN : All of us know that the natural course of acute tubular necrosis passes through three clinically recognised phases. First, we have oliguric phase which may last one or two weeks, followed by the phase of diuresis with abrupt increase of urine volume and often progressive polyuria, and finally the post diuretic phase of gradual recovery of kidney function. Gp Capt Krishnamurthy will agree that during the oliguric phase there is retention not only of water but also of urea, potassium and acids, due to uncontrolled endogenous protein catabolism. Which of these is of serious clinical significance ?

GP CAPT KRISHNAMURTHY : Hyperkalaemia is possibly the most important biochemical change because it leads to cardiac irregularities if the level is allowed to rise beyond 7 mEq/L. Overhydration often complicates matters, especially in cases receiving earlier transfusions freely, leading to pulmonary oedema or congestive failure. Rising blood urea often produces gastrointestinal symptoms of vomiting, diarrhoea and mucosal bleeding. Deepening coma sets in when the blood urea level approaches 400 mgm/100 cc.

CHAIRMAN : It is not the actual level but the rate of rise that really matters. If the blood urea rises at the rate of 40 to 50 mgm a day, even a lower level may cause serious symptoms. Col Vasudevan, is the clinical and biochemical picture in any way different in chronic renal failure ?

LT COL VASUDEVAN : In chronic renal failure the clinical picture is dominated by polyuria, hypertension, osteodystrophy, anaemia and azotaemia. The danger of hyperkalaemia is not so acute unless acute renal failure supervenes on chronic renal failure. A much higher level of blood urea may be tolerated in these cases without any clinical manifestation.

CHAIRMAN : Col Yoginder Singh, we have already pointed out that for proper planning of treatment, it is essential to distinguish functional failure from acute tubular necrosis. But how should one go about it, in actual practice, without depending too much on laboratory reports ?

COL Y SINGH : First we must correct hypovolaemia with transfusion of appropriate fluids like electrolyte solution, plasma or blood. C.V.P. monitoring should be done to prevent over-transfusion. If oliguria persists, 200 cc of 10% Mannitol I.V. may be given in 10 minutes If the urine flow increases to more than 40 cc/hour, more Mannitol is indicated to maintain the urinary output. Lack of response to Mannitol indicates acute tubular necrosis, and appropriate restriction of further fluid transfusion. Frusomide 200 mgm I.V. may also be used. It should produce three-fold increase in urine output in 5 to 30 minutes. This is actually safer than Mannitol, which if ineffective, may lead to fluid overload.

CHAIRMAN : Lt Col Khanna, since we cannot do anything about the progress of the tubular necrosis, how are we going to sustain the patient ?

LT COL KHANNA : In addition to the restriction of water and electrolytes one must provide 2,000 calories daily in the form of carbohydrate by the oral route, or if the patient is vomiting, by I.V. route, in the form of concentrated glucose using polythene tubing introduced into a large vein such as inferior vena cava. So long as enough carbohydrate is available, patient may be given 20 to 30 g of protein of high biological value like egg or cheese to cut down protein catabolism or periodic blood transfusion.

CHAIRMAN : Lt Col Sharma, it has been mentioned that hyperkalaemia is a dangerous complication of acute renal failure. How should one manage the patient in the face of rising serum potassium ?

LT COL SHARMA : The treatment should consist of :—

(a) I.V. hypertonic glucose as already suggested but combined with 20 units of soluble insulin.

(b) 10% calcium gluconate, 20 ml I.V.

(c) Sodium bicarb 50 to 100 mEq I.V.

All these may be repeated as and when necessary, but one should be careful about the total fluid and sodium intake. Ion-exchange resins are not popular these days. The ultimate answer, of course, lies in dialysis.

GP CAPT KRISHNAMURTHY : DG's Memorandum on Renal Failure recommends prompt digitilization as the first step in the management of hyperkalaemia. But this is better avoided if there is no incipient congestive heart failure, even then it requires caution.

CHAIRMAN : When kidneys are functioning, one has to be cautious in using drugs which may not be excreted by the kidneys. What are the drugs one must be specifically careful in using ?

GP CAPT KRISHNAMURTHY : Drugs like phenobarb, digoxin and antibiotics of penicillin group should be reduced and tetracycline which is nephrotoxic, should also be avoided.

CHAIRMAN : But I suppose if the patient is being dialysed, there is no problem of drug accumulation unless the drug is bound to plasma protein. Lt Col Khanna, is there any place for active surgical intervention in cases of acute renal failure ?

LT COL KHANNA : If it is a case of concealed accidental haemorrhage, early evacuation of the uterus by rupturing the membranes and pitocin drip is advisable. In septic abortion one should be more conservative. If it is due to eclampsia, then hastening the delivery will be justified.

GP CAPT KRISHNAMURTHY : I don't know if the use of pitocin is all that safe. As far as we know, it might aggravate renal vaso-constriction.

LT COL KHANNA : Well, now we are between the devil and the deep sea. The foetus has to be got rid of. The only answer is a caesserian. But it has to be carefully thought of in view of the shock.

CHAIRMAN : There is no end of controversy when physicians and surgeons confront each other. Let us not forget that there is scope for surgical intervention even in non-pregnant females as well as the males suffering from acute renal failure. The role of debridement and amputation of gangrenous limb may be life-saving in certain cases of battle injury with acute renal failure. There are reports of acute renal failure following attempted reimplantation of severed limbs. One should also know when to sacrifice the limb to save a life. Major Kumar may let us know the scope of surgical intervention in chronic renal failure.

MAJOR KUMAR : In the presence of severe uraemia, patient may need preoperative dialysis to make the surgery safe. Dialysis may also permit a high dose I.V.

pyelography in cases where diagnosis remains uncertain. Depending upon the nature of obstruction, various operations can be carried out to correct obstructive uropathy.

CHAIRMAN : Col Yoginder Singh, could you tell us when should we really start thinking of dialysis ?

COL Y SINGH : Dialysis should be used as an adjunct to conservative management. The indications are—hyperkalaemia of 7 mEq/L, blood urea 300 mgm% and CO_2 combining power less than 12 mEq/L. The trend is to dialyse early to forestall uraemic complications. We should be guided more by the clinical picture rather than the laboratory reports.

CHAIRMAN : Lt Col Vasudevan, you have a lot of experience with peritoneal dialysis. Could you tell us the usual technique one should follow ?

LT COL VASUDEVAN : After emptying the bladder, a single polythene catheter with multiple holes should be introduced aseptically into the peritoneal cavity, through a ub-umbilical or iliac puncture. 2 litres of sterile dialysing fluid, which normally consists of 1.5% dextrose, and electrolytes similar to plasma should be run in and then siphoned out after 30 to 40 minutes. This procedure should be repeated 10 to 20 times a day with frequent check-up of clinical and biochemical status.

CHAIRMAN : Is there any scope or indication for changing the composition of the dialysing fluid ?

LT COL VASUDEVAN : When serum potassium is high, the fluid should be potassium free. 5% dextrose may be necessary to remove the excessive fluid from the body. Heparin and antibiotics may be added to the blood though it should not be used as a routine.

CHAIRMAN : Col Yoginder Singh, you must have had some occasions to carry out periotoneal dialysis. Did you have any problems ?

COL YOGINDER SINGH : The main problem is blockage of the tube by fibrin. Another problem is leakage of fluid from the recent laparotomy wounds. The intraperitoneal adhesions often give trouble during introduction of the tube. Extensive burns often create problems of sepsis.

CHAIRMAN : Lt Col Sharma, is haemodialysis a quicker and more efficient method of managing cases of renal failure ?

LT COL SHARMA : Haemodialysis is no doubt a quicker and more efficient method, but it is more useful in chronic renal failure. Haemodialysis programme can keep the patient with end-stage kidney alive for an indefinite period. Haemodialysing units are available which can be operated by patients' relations with minimal training. Treatment of chronic uraemia by haemodialysis has been made easier by the development of Schribner's external arterio-venous shunts of sialistic tubing or Bresica arterio-venous fistula, which allows puncture without any danger. However, patients in acute renal failure in a hypercatabolic state may need haemodialysis where other methods have failed.

CHAIRMAN : Lt Col Sharma, you have said that patients with chronic failure can be maintained indefinitely on haemodialysis. Are there any chances of a complication ?

LT COL SHARMA : There are problems like shunt failure, bleeding and hepatitis. Most of these shunt problems have been solved by introduction of AV fistula where arterialised vein can be punctured repeatedly without any risk of infection or blockage. Bleeding tendencies have been tackled by regional heparinisation and viral hepatitis by minimising blood transfusion and by screening the blood for Australia antigen.

CHAIRMAN : What about surgery and haemodialysis ?

MAJOR KUMAR : Apart from any surgical emergencies which may arise in a patient, bilateral nephrectomy may have to be performed prior to transplant for controlling intractable hypertension and the patient will require haemodialysis a day before surgery. During the post-operative period, dialysis should always be avoided for the first 48 hours.

CHAIRMAN : Lt Col Vasudevan, in acute renal failure if the kidneys fail to recover their function, how long one should persist with dialysis ?

LT COL VASUDEVAN : Our experience is very limited. The longest period for which peritoneal dialysis has been carried out for acute renal failure is 8 days. If a patient remains oliguric for over 3 weeks, then one must suspect the possibility of cortical necrosis. There is a place here for renal biopsy. Once cortical necrosis is confirmed, one has to decide whether the patient should be left alone or taken up for intermittent haemodialysis and subsequent renal transplantation.

CHAIRMAN : Our experience with the Kolff's artificial kidney with its big rotating drum has not been very encouraging. But let us remember that for acute renal failure, peritoneal dialysis is the method of choice because it can be initiated in any hospital set up without trained personnel and expensive equipment.

CHAIRMAN : Major Kumar, What is the scope and limitation of a kidney transplant ?

MAJOR KUMAR : Lt Col Sharma has already pointed out that patients with end-stage kidneys can be kept alive indefinitely on haemodialysis. But this is not the answer, because apart from the cost the patient has to depend upon the machine, restricting his travel. The answer lies in renal transplant. The best chances are with kidneys taken from monozygotic twins, if available, or a closely related donor. The donor, besides being of the same A B O blood groups, the tissue match should be satisfactory in respect of H L A tissue type and mixed lymphocytic culture. Even then, patient will need immuno-suppressive drugs like Imuran and steroids. There is general trend to use cadaver kidneys, though the results are not as good as with closely related living donor kidneys. In our country we have also the legal problems in respect of cadaver kidneys which need to be solved.

CHAIRMAN : Let us not forget that like heart transplant, there is also an ethical aspect of the problem of renal transplant. Since the best chances of survival are with kidneys from closely related living donor, we have to ensure that the prospective donor, be it father, mother, brother or a sister, is protected against undue pressure from the patient, his family or his own conscience. The donor must consent without pressure. Even when using cadaver graft, we should make sure that the patient being maintained well on intermittent haemodialysis is not made worse after the kidney transplant and long continued immuno-suppressive drugs.

And now winding up the discussion, I want to emphasise that acute renal failure is usually an emergency arising in the context of surgical, obstetrical or medical shock from which recovery is possible, provided a temporary alternative arrangement is available to take over the functions of the kidneys for the period, they decide to remain in a state of complete shut down. In practical terms this means dialysis usually peritoneal, unless the breakdown of body protein is so rapid that only haemodialysis can cope with the products of protein metabolism.

Chronic renal failure, on the other hand, is usually a long drawn illness whose management may extend over many years. In all cases of chronic renal failure attention must be paid to the possible existence of a remediable obstruction of the urinary tract. In most cases, however, by the time chronic renal failure has produced symptoms of uraemia, the process of renal destruction is already far advanced and haemodialysis followed by a transplant programme has to be seriously considered.

I thank all members of the panel for their excellent cooperation and the distinguished audience for their keen participation in the proceedings of this panel discussion, I will now request Lt Gen Barat, our Chief Consultant in Surgery to say a few words about the first renal transplant in the Army Hospital done recently.

LT GEN AK BARAT : I congratulate Brig Ganguly and members of the panel for their lucid and critical presentation of the various problems in the management of Renal Failure.

As regards the immediate management, the important points brought out by individual speakers are generally accepted and there is little room for further elaboration. In cases of chronic renal failure, intermittent haemodialysis followed by renal transplant is the answer.

As regards the first ever kidney transplantation carried out in the Army Hospital in November 73, we have to admit that we have yet to catch up with the accepted routine that is in vogue not only abroad but also in some of the centres in India, of which Christian Medical College and Hospital, Vellore, needs a special mention.

In the Army Hospital, we carried out the first kidney transplantation in the Armed Forces for an end-stage kidney disease in a young officer who needed haemodialysis almost once a week. After proper tissue matching, a living donor came forward to donate a kidney voluntarily to save the patient's life, which was gratefully accepted. The transplantation was carried out in the twin theatre of Army Hospital, with two teams working simultaneously. All steps of the operation went according to plan and the accepted technique was adopted without any flaw. The patient was apparently doing well on the 7th day, though the blood urea had gone up in spite of good urinary output. However, in view of the rising blood urea, the transplantation team decided to undertake one haemodialysis to tide him over the crucial period, though the creatinine and potassium level was not very high. The patient was on heavy dosage of immuran and corticosteroids as well as antibiotics and was apparently doing well after haemodialysis. But soon after the haemodialysis he started having severe haematemesis and malaena which did not stop in spite of repeated blood transfusion and other supportive treatment. The patient went into irreversible shock and died. Post-mortem showed gross ulceration of the mucosa at the cardio-oesophageal junction and mucosal erosion of the stomach as well as the whole of the colon. Uncontrollable haemorrhage is known to be a complication following renal transplantation. We wonder if immuno-suppressive drugs are double edged weapons, which were responsible for the breakdown of the barrier between infection and the mucosal resistance, especially in the colon. Uraemic ulcers are also known to occur in renal failure, and we do not know if this was precipitated by the immuno-suppressive drugs and haemodialysis.

This was our first case and we feel very sad about losing the patient though the transplant was successful and subsequent histopathology did not show any evidence of rejection. We have learnt some lessons and we will pursue the renal transplant programme in future with a hope of success.

OPEN HEART SURGERY IN THE ARMED FORCES

BY

Colonel A. M. Ahuja*

There has been rapid progress in Medicine in the last 2-3 decades, but in the case of Cardiac Surgery the progress has been most fantastic. It is only 20 years back that Dr. Gibbon at the Mayo Clinic successfully tried out his extra-corporeal machine. Since then every few months, fresh advances are occurring to cover an ever widening field in cardiac pathology.

It was in this context that in 1966, it was realized that although the Armed Forces were the very first in the country to establish a centre exclusively for thoracic surgery at M.H. Aundh in 1942, this centre was not doing enough cardiac surgery, especially open heart surgery, to be able to compete with similar civilian centres who had made rapid strides while we waited. It was also realized that the congenital cardiac defects or seriously diseased valves were no longer the domain of fatalistic philosophy or pathological curiosity and that open heart surgery was an important advance which must be practiced by a self-respecting thoracic surgical centre. At the same time it was obvious that sending our service personnel or their dependents to civilian centres would be costly, inconvenient and an unsatisfactory solution.

Based on this requirement, a few basic machines and a research recorder were imported and the team started a series of experiments on dogs to perfect the drills and routines required to ensure satisfactory extra-corporeal circulation. Three surgeons, 2 anaesthetists and 2 nursing officers were sent to well known centres to pick up the special know how. Successful open heart surgery commenced in 1970, but the programme was hampered by difficulty in getting a continuous supply of equipment much of which is imported. This difficulty has now been removed for which we are thankful to our DG AFMS, Lt. Gen. Ved Parkash, PVSM, who has streamlined the supply procedure. We are also grateful to our Chief Consultant, Lt. Gen. A K Barat, PVSM, MVC with whose help, guidance and encouragement the open heart surgery is now a routine affair.

The aim of this paper is to present a resume of our work in the last 3½ years, a birdseye view of how this team functions, and a word about the future programme of this centre.

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RESUME OF WORK

Upto date 53 cases had open heart surgery, the pathology of which is classified in table I. The mortality figures are given in brackets.

Table : 1—Resume of work

(Figures in brackets show mortality)

ASD Secundum	—	22	(1)
ASD Sinus Venosus with Anomalous Venous Drainage	—	6	
ASD Primum	—	1	(1)
Pulmonary Stenosis	—	5	
Tetralogy of Fallot	—	4	(2)
Cor Triatriatum	—	1	(1)
Triology of Fallot	—	2	
Ventricular Septal Defect	—	6	(2)
Mitral Commissurotomy with L A Clot	—	1	
Mitral valve Replacement	—	3	
Atrial Myxomas	—	2	
	Total	53	(7)

RESULTS

Out of 53 patients, 46 survived the operation to have uniformly good to excellent results. They had smooth post-operative course and there were no late complications or deaths.

The 7 deaths occurred in the beginning of our series. Most of them are today preventable as more experience has been gained. The solitary death in the ASD secundum series was due to cerebral air embolism. The case of Cor Triatriatum, a very rare pathology, was not correctly diagnosed during the early period when our cardio-angiography was not functioning. The total correction of Tetralogy of Fallot is a high mortality field (2 out of 4) and so is the repair of VSD with pulmonary hypertension (2 out of 6). With added experience we hope to cut down our mortality in these 2 types of poor risk cases.

TEAM WORK

In comparison to other branches of surgery, the open heart surgery team consists of a much larger number of essential members, every one of whom must do his part reliably and perfectly to ensure success. Failure of any one of them has a heavy penalty i.e. the death of the patient. The common saying "A chain is as strong as its weakest link" is very apt in this set up.

Let me trace a patient who has open heart surgery.

Based on clinical, X-Ray and ECG findings, a physician makes a provisional diagnosis of a correctable defect. This patient is then studied by cardiac catheterisation and angiocardiography to outline the exact location and type of the defect or combination of defects.

A correct diagnosis before operation is of paramount importance. It is not possible to explore all chambers at surgery. The limited time that a surgeon has when the patient is on extra-corporeal machine permits only a pre-planned operation. A wrong anatomical diagnosis means a wrong operation and this generally means the death of the patient. Hence the physician incharge must have proper equipment and experience to produce a diagnosis which is always confirmed at surgery.

Once the surgical team takes over the case, the surgeon depends upon the team of anaesthestists to :

(a) administer safely a prolonged anaesthesia.

(b) run all the open heart surgery machines, the ECG and pressure monitors and biochemistry apparatus simultaneously and faultlessly for the life of the patient totally depends upon machines during the period of cardiopulmonary by-pass.

The surgeon and his assistants must do their part accurately and with finesse. They should recognize the pathological anatomy of the defect and be able to define the vital and dangerous areas in the jungle of fine anatomical structures inside the heart, so that the final repair is accurate. The price for unsatisfactory repair again as a rule is death of the patient.

At this stage I must add the important role of the blood transfusion department who must give us thoroughly matched, aseptically drawn blood in large amount.

After surgery the patient must move to the ICU where close observation, continued electrical monitoring and frequent biochemical estimations must continue until the patient is stabilized for routine care. Any complication that occurs must be immediately recognized and counteracted. In short to carry a patient successfully through open heart surgery means long operating and many sleepless nights for the team until the patient can be discharged for routine follow up by the physician concerned.

FUTURE PROGRESS

The capacity to perform successful open heart surgery is an important addition to our treatment facilities. The centre, in a short time, hopes to perfect coronary angiographic techniques and be able to bypass diseased and obstructed coronary arteries by saphenous vein grafts and thus be able to help selected patients who have persistent angina or threatened recurrence of myocardial infraction.

ACKNOWLEDGEMENT

I am grateful to each and every member of the team who has made the success of this programme possible. My special thanks are to the anaesthesia team i.e. Wg Cdr S K Bhalla, Lt Col S P Mallick, Major S Mandal, and Colonel S Thirumalai who was with us in the initial and more difficult pioneering period. My thanks are to our solitary cardiologist Lt Col I M Ahuja, to the BTD of AFMC, to our pathology and X-Ray departments. The main brunt of surgery and post-operative care was shouldered by the surgeons of the team i.e. Wg Cdr H M Aga, Lt Col R N Thareja and last but not the least, by Major V Ramachandran. Among the nursing officers our O.T. incharge matron Major (Miss) J Vaz needs a special mention.

MICROSTUDY AND MICROSURGERY OF THE EAR

BY

Brig Daryao Singh

Like other branches of medicine, Otolaryngology has advanced from the days of guillotine surgery to the present era of microsurgery. Aural Surgery has now become more precise and new fields have opened up in the treatment of aural disease. Treatment of middle ear infections has undergone a radical change. Treatment of ostosclerosis is now no more disappointing. Facial nerve [1] can now be repaired and restored to normal function. Menieres disease [2] can be treated by surgery on the saccus endolymphaticus in the posterior cranial fossa. Acoustic nerve tumours[3] can be completely removed in their early stages through the aural route. Even the minutest details of the cochlea can now be studied through specialised microtechniques e.g. scanning [4] electron microscopy.

Attempt will be made here to refer to some of the advances in microsurgery of the ear particularly relevant to the Armed Forces.

I. Tympanoplasty : C.S.O.M has been the commonest disease of the ear and the aural surgeon has always been preoccupied with controlling infections of the middle ear tract and treating its complications which can be quite varied and very serious. The middle ear cleft, is situated adjacent to the intracranial structures. Infections of the middle ear are, therefore, a source of potential danger to the brain or the meninges. Thus, extradural abscess, meningitis, brain abscess and lateral sinus thrombosis may arise from extension of infection from the middle ear and mastoid. A special feature of he middle ear infection is the formation of cholesteatoma which erodes bone and facilitates the spread of infection.

Liability of serious, and quite often fatal, complications arising from infection of he middle ear has always been the dread of ENT surgeons of preantibiotic era. Their whole energy had been concentrated on eradicating infection without much concern about the function of the ear itself. Stress was on controlling mortality rather than on preventing morbidity. Radical operations were carried out on the ear without regard to he sound conducting mechanism. Of course, no attempt was made to reconstruct the middle ear mechanism which had been destroyed by disease.

With the advent of the operating microscope, it is now possible to perform precise urgical techniques and to reconstruct the hearing apparatus. The operation is broadly named as Tympanoplasty. The principles of this operation include : (1) Reconstruction of tympanic membrane (2) Reconstruction of ossicular chain and (3) conservation of all healthy tissue and preservation of the normal anatomical pattern of the ear.

The tympanic membrane is made up of a middle fibrous layer covered on the outside by skin continuous with the skin of external auditory meatus and on the inside by mucous membrane continuous with the mucosa of the middle ear. In the operation of Tympanoplasty, the middle fibrous layer is reconstructed by means of temporal facia graft. The skin and mucous layer are formed later by the gradual growth of these tissues from the adjacent parts.

The ossicular chain is reconstituted either by homograft ossicles or by means of polythene or teflon prosthesis.

Healthy tissues are strictly conserved and only the diseased tissues are removed by precise dissection under the microscope. It is important to preserve or to reconstruct as far as possible, the normal anatomy of the various parts of the external and middle ear.

In the armed forces, tympanoplasty is particularly relevant to reconstruct the drumhead and the middle ear disrupted through trauma, during peace or during war. A number of cases [5] sustained such injuries during Indo-Pak war.

II. Stapes Surgery : Stapes bone has come to acquire unique importance in otology because of its selective involvement by a disease called—OTOSCLEROSIS. Nobody exactly knows why this happens. Aetiology of this disease is still in the realm of guessing and postulating various theories. This disease assumes a greater importance because it affects people when they are young and because it is progressive in nature, affecting both ears at the same time and, in time, leading to gross deafness. We in the armed forces deal mainly with this age group and hence the importance of this disease. Out of 222 cases of Otosclerosis[6] treated at Army Hospital, DELHI CANTT, 103 cases were between the age of 19 and 30 years.

Otosclerosis, like Pagets' disease, causes softening of bone followed by sclerosis. As a result of this pathology, the stapes bone starts losing its normal mobility and ultimately becomes completely fixed in the fossa ovalis. As the normal conduction of sound in the ear depends upon the mobility of the ossicular chain, fixation of stapes leads to a conductive type of deafness. It means that the patient is deaf, though his sound perceiving mechanism in the inner ear is quite healthy. In a few cases, the disease spreads deeper and involves the inner ear as well, giving rise to a mixed type of deafness.

Stapes is a small bone, deeply situated in the ditch of the fossa ovalis. Its details are hardly visible to the naked eye and surgery on this bone had not been possible before the coming of the microscope. The microscope has now made this precise surgical procedure possible. When the operating microscope was first introduced in the forties, surgery for the relief of otosclerotic deafness was perfomed well away from the stapes by making a new opening in the lateral semicircular canal which was relatively easier to approach. This operation was called fenestration operation by Lempert. But this operation had its limitations because the benefit of the lever mechanism of the ossicular chain was lost. Hearing gain was therefore limited. Also, the new window tended to close through nature's attempt to restore the normal anatomy.

The modern operation for otosclerosis is a direct attack on the stapes bone, the site of the disease. The principle of the operation comprises removal of the stapes and its replacement by a prosthesis. The operation is called STAPEDECTOMY. The stapes is removed and the opening of fossa ovalis is covered with vein graft. Gap between the incus and this vein graft is reconstituted by means of a teflon piston.

As a result of Stapedectomy, 95% of all suitable cases of otosclerosis regain socially adequate hearing.

III. Microstudy of the Cochlea : The above mentioned two operative procedures were concerned with the surgery of the middle ear or the conducting mechanism of the function of hearing. Microscope has opened up the dark and narrow recesses of the inner ear labyrinth and has brought the study of microstructure of cochlea within the scope of the E.N.T. Specialist.

In the armed forces, acoustic and blast trauma to the inner ear are of special interest to us. Troops are exposed to the hazards of noise in the army, in the navy and in the air force. The level of noise goes as high as 180-190 Decibels, whereas exposure to a noise level of more than 80 Decibels for any length of time is known to give rise to permanent deafness. Then, there is the hazard of blast trauma due to exposure to still higher pressures of bomb and shell bursts near the troops during war.

During the two Indo-Pak wars we studied 133 cases of blast injuries of the ear. The troops had sustained all type of deafness : conductive, perceptive or mixed i.e. involving the middle ear, the inner ear or both. There were cases of perforation of tympanic membrane, dislocation of ossicles and haemorrhages in the inner ear.

While studying these cases of blast deafness, we noticed that the turban worn by Sikh troops protected their ears against blast injuries. We therefore carried out laboratory studies using various type of material to study their protective action against blast. We found that a baffle of wire mesh and foam[7] effectively occluded blast effects without affecting the hearing. Different thickness of foam provided variable attenuation and maximum thickness of foam provided an attenuation of 90% of Intensity of blast as shown in the Table I. This finding was of great potential benefit because we could devise a protector against blast without hampering communication in troops during action.

TABLE I

Sample No.	Material	Sound attenuation in lb. (at threshold level)										Shock attenuation percentage		
		125 Hz.	250 Hz.	500 Hz.	1,000 Hz.	1,500 Hz.	2,000 Hz.	3,000 Hz.	4,000 Hz.	6,000 Hz.	8,000 Hz	1st Set	2nd Set	3rd Set
1.	2 mm. foam in muslin	5.0	5.5	4.5	3.0	1.0	1.0	1.0	0	0	0	45	52	51
2.	3 mm. foam in muslin	9.5	3.5	6.0	3.0	2.5	1.0	1.0	1.0	0.5	0.5	62	63	68
3.	2 mm. foam in nylon net	2.5	3.5	4.0	2.0	1.0	1.0	2.0	0.5	0.5	0.5	47	41	44
4.	3 mm. foam in nylon net	3.5	2.0	1.5	1.5	3.0	4.0	3.5	2.0	1.5	1.0	50	59	57
5.	2 mm. foam in cotton net	1.5	1.0	2.5	2.0	3.0	1.0	0	0	0.5	1.5	60	50	61
6.	3 mm. foam in cotton net	2.0	2.0	1.5	2.5	2.5	3.0	5.0	5.3	2.5	1.5	65	63	66
7.	2 mm. foam in 50 wire mesh	4.0	4.5	3.5	2.0	0	0	1.5	3.5	4.0	2.0	69	71	72
8.	3 mm. foam in 50 wire mesh	4.0	4.5	3.5	3.0	2.5	3.0	6.0	5.0	5.5	4.0	89	92	92
9.	2 mm. foam in 30 wire mesh	3.0	3.5	5.5	3.0	3.0	2.0	1.0	1.0	0	0	71	70	70
10.	3 mm. foam in 30 wire mesh	4.0	4.0	3.0	3.0	2.0	1.0	1.0	1.5	1.5	1.5	89	88	91
11.	2 mm. foam in 25 wire mesh	2.0	3.5	4.5	2.0	3.0	3.0	5.0	4.0	2.0	2.0	84	70	70
12.	3 mm. foam in 25 wire mesh	1.5	6.0	4.5	5.0	2.5	3.5	3.0	2.0	2.0	1.5	86	87	85
13.	2 mm. foam in 18 wire mesh	2.0	1.5	1.5	3.0	2.5	3.0	1.5	2.0	2.0	2.0	59	62	62
14.	3 mm. foam in 18 wire mesh	2.5	4.0	4.0	2.0	1.5	1.0	1.0	0	0	1.0	89	87	80

Again we studied these blast protectors[8] in Guinea Pigs. Here also these gave full protection against Blast Pressures, while Guinea Pigs without these Protectors developed rupture of tympanic membrane and gross deafness, those with blast defenders placed over their ears (Fig 1), had no injury to their ears.

We are also studying the effects of blast on inner ears in Guinea Pigs because from the study of battle casualties we feel that the nature of inner ear lesions would be different than the type of damage observed in noise induced deafness.

The Guinea Pigs are exposed to a measured amount of Blast Pressure at a Defence Science Laboratory. Initially animals were exposed to a Blast Pressure of 5—20 lbs per square inch. This resulted in rupture of DRUM MEMBRANES and deafness. No damage was seen in the inner ear (Fig 2) when examined by surface technique[9] although in some animals haemorrhage was seen in the inner ear when examined by light microscopy.

It was decided to reduce the blast pressure to find out the pressure at which drum membranes do not rupture, but animals still become deaf, as it appeared that rupture of Tympanic membrane gave protection to the inner ear. It was observed that at 1-2 lbs per square inch pressure, tympanic membranes do not rupture but animals definitely develop deafness.

It is now proposed to expose at least 20 Guinea Pigs to a blast pressure of 1—2 lbs per square inch and at least give 4 exposures to half of these animals and then examine the inner ear.

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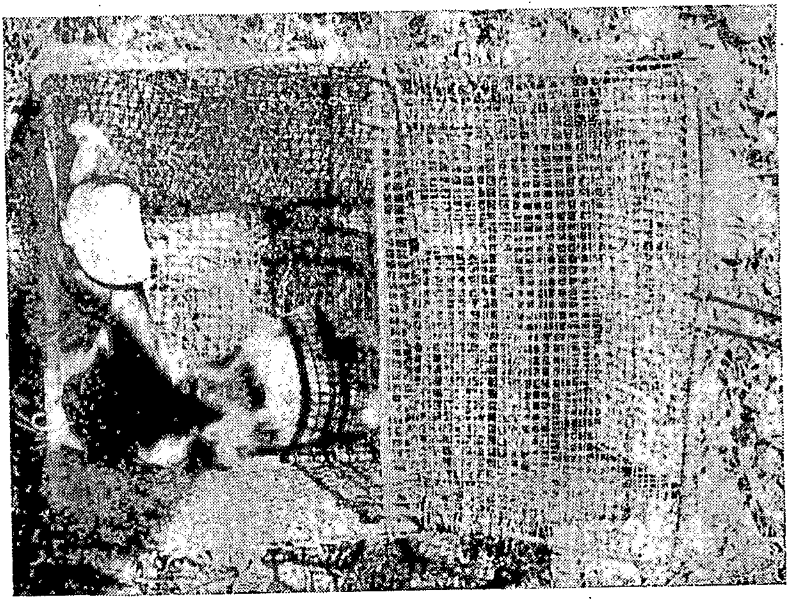

Fig. 1—Guinea Pigs with blast defenders

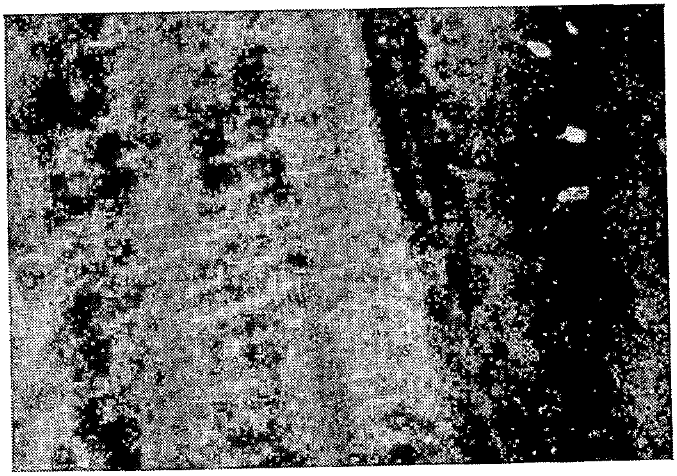

Fig. 2—Inner ear examined by surface technique

KERATOPALSTY

BY

Col G. S. Sihota

Corneal grafting is a well established and a widely practised procedure. In keratoplasty alone among the various fields of surgery, we have functionally successful homotransplants of a permanent type in a high percentage of our patients.

A beginning has been made in this procedure in the Armed Forces and 21 cases have so far been done at Army Hospital.

Indications

Following are the main indications:—

(i) Keratoconus, corneal dystrophies, blood staining of cornea and inactive interstitial keratitis.

(ii) Corneal opacities

  (a) Small central opacities with no vascularization

  (b) Small opacities which are eccentric and vascularized

  (c) Large heavily vascularized opacities.

(iii) Herpetic/indolent ulcers

(iv) Multiple corneal Foreign bodies

(v) Malignant recurrent pterygium

Types of Corneal grafts

(a) Penetrating (full thickness)

(b) Lamellar (Partial thickness)

  (i) optical

  (ii) Preparatory

  (iii) Therapeutic

Scope in Armed Forces :

There is enough scope for this microsurgical procedure as we encounter every type of ocular disease among soldiers and their families. Only the number of cases will be less as compared to the civil.

Of special interest are the unilateral cases of corneal disease with the other eye normal. Such cases are usually left alone in the civil due mainly to paucity of donor material. But in the Armed Forces, other ranks with marked diminution of vision in one eye are placed in a permanent low medical category. These personnel are sent on release by the Regimental centre. This besides resulting in the loss of a trained soldier also renders the person jobless.

We have done Keratoplasty in such cases with a view to upgrade them to medical category Aye and have succeeded in doing so.

DONOR MATERIAL

Source

Eye ball is removed under aseptic precautions from a dead body within 6 hours of death and stored at 4°C in sterile liquid paraffin. This cornea should be utilized as early as possible although it is known to give good results upto 48 hours.

Army Hospital is recognised under Corneal Grafting Act of Delhi and an eye can be removed for grafting purposes from a dead body with the permission of near relatives. In the absence of near relatives, officer Commanding Hospital is authorised to give such permission. We have so far procured our donor corneas from dead bodies of persons whose near relatives were not present in the station.

As eyes are used within 48 hours of removal, elaborate arrangements for an Eye Bank are not required and domestic refrigerator suffices.

As and when supply of donor cornea from Army Hospital falls short of the requirement, it may be possible to get more donor material from a hospital like 92 Base Hospital. This hospital is considered suitable because of the following factors :—

(i) Regular daily air service.

(ii) Being in a field zone, there will be no near relatives in the majority of cases.

(iii) Special thermos flasks have been procured which when packed with ice maintain temp of 4°C for 48—72 hours.

We cannot depend on the civil for the donor material as they can hardly meet their own requirement. We will thus have to be self-sufficient in donor material.

Analysis of cases done at Army Hospital

Category of patients

(a) Serving Personnel	16
(b) Families	5

Types of Grafts

(a) Penetrating (full thickness)	14
(i) Optical	11
(ii) Structural	3
(b) Lemellar (Partial thickness)	7
(i) Optical	1
(ii) Preparatory	2
(iii) Recurrent pterygium	4

Disease/disability :

(a) Corneal opacity	13
(b) Keratoglobus	1
(c) Recurrent pterygium	4
(d) Perforated Corneal ulcer	3

Size of Grafts (Penetrating)

6 mm	1
6.5 mm	8
7 mm	3
7.5 mm	1
8 mm	1

Results

(a) Penetrating (Optical)	11 cases
(i) Good visual result	5
(ii) Graft reaction (failure)	1
(iii) Lost to follow up	2
(iv) Not reached finality	3
(b) Penetrating (in perforated ulcers)	3 cases

All three were successful in the sense that the eyes were saved which otherwise would have been enucleated.

(c) Lemellar (Optical) 1

This was successful—vision improved from perception of light to 6/36 in a squinting eye with nystagmus.

(d) Lemellar (Preparatory) 2

In one vision improved from perception of light to 6/24. This housewife was extremely happy as she was virtually blind in both eyes.

The other case in still in hospital

(e) Lemellar (Recurrent pterygium) 4

All four cases were successful.

Graft Reaction

We are fortunate that homograft reaction does not occur so frequently in keratoplasty but the problem is there and leads to opacification and vascularisation of the graft. It can neither be foreseen or prevented nor can it be really cured. We depend on non-specific reaction-suppressing agents like local and systemic use of corticosteriods as well as Beta Rays Thio-tepa and Mitomycin have also been used as eye drops. Potent anti-metabolites are not in vogue in keratoplasty.

MODERN CONCEPTS IN THE MANAGEMENT OF HEPATIC INJURIES

BY

***Lt Col H. C. Puri, VSM, FRCS, FRCS E.**

The "Jet" age has produced a marked upsurge in the number of blunt and penetrating injuries to the abdomen. These injuries result from stab wounds by sharp implements or fire-arms, but more so, by the rim of the "recessed" central post of steering "wheel", or seat belts worn across the lower abdomen. Such trauma, produces a wide range of injuries—from mere contusion of the abdominal wall to massive and multiple lacerations of the abdominal organs. Although, the indications for exploratory laparotomy may be evident in the more severely injured, the proper diagnosis and management of these patients are less well defined. The clinical picture at times is so confusing, to tax the skill of the most experienced clinicians.

The recent discoveries regarding the anatomy of the liver and a more complete physiological understanding of the physiological functions of the liver, has made it possible not only to diagnose, but also, to treat hepatic trauma successfully, in the majority of the cases. This was imperative as classic methods of treatment of severe lesions of the liver were found to be inadequate.

Anatomy of the liver

The liver is now considered to be divided anatomically into Rt and Lt halves, by the principal plane from the north of the gall bladder fossa to the inferior vena cava fossa. (Fig. I & II). The central vein lies along this principal plane which divides the liver anatomically into Rt and Lt halves. The variations of this hepatic venous system are thus vital in major liver resections, and have been very ably described by M Balasegram. The falciform ligament divides the left anatomical half into medial and lateral segments. Classically, left lobectomy requires resection of the left lobe to the left of the falciform ligament, while in left hemi-hepatectomy the liver is resected to the right of the falciform ligament. Since the central vein lies along the principal plane and drains part of both right and left anatomic lobes, right or left hemihepatectomy must be a little to one or the other side of the plane. Extended Rt or Lt hemihepatectomy involves removal of each respective anatomic lobe drained by the central vein. Further though

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surgically unfeasible the Rt anatomical lobe has also been divided into posteral-lateral and antero-medial segments (Fig. III).

Pathology of hepatic trauma

(a) Rupture of the liver with laceration of Glisson's capsule.

(b) Separation of the capsule with sub-capsular haematoma.

(c) Central rupture with hematoma, abscess, or bile cyst.

Penetrating injuries

Penetrating injuries vary from clean incisions produced by knife wounds to large areas of liver trauma secondary to gunshot wounds. The magnitude of injury which accompanies a missile wound is determined by the explosive effect of the missile, rather than its size. The wound of entrance is usually smaller than the wound of exit, and tissue involvement extends out circumferentially from the path of the projectile. Damage to blood vessels and the pressure or intraparenchymal haemorrhage produce further devitalization and necrosis. In penetrating lesions of the lower part or the thorax, the dome of the liver is most frequently involved, and the ratio or right lobe to left lobe injury is approximately 7 : 1. With penetration of the anterior abdominal wall, the anterior surface of the liver is usually affected. The main hepatic vessels and bile ducts are involved only infrequently, because of their protected position on the inferior surface of the liver.

Nonpenetrating Injuries

Nonpenetrating injuries are caused by kicks, falls, and compressive trauma to the lower chest or upper abdomen which frequently accompanies automobile accidents. The injury varies from simple capsular tear, with relatively minor interruption of the underlying liver parenchyma, to gross cleavage of the organ. When a compressive force is applied, the liver tears in an anteroposterior direction. When extreme flexion is implicated, the direction of tear is usually transverse. The dome of the liver is the part most frequently ruptured in older persons, and cracks in the sagittal planes are commonly found on the upper convex surface. These may appear as shallow fissures, more or less parallel with one another, or as a single large laceration which divides the parenchyma partially. In extreme cases, large fragments of the liver or entire lobes may be detached and floating free in a pool of blood in the peritoneal cavity.

When rupture of the liver extends through Glisson's capsule, extravasted blood and bile may be found in the peritoneal cavity, and with associated wounds of the diaphragm, the fluid may find access to the pleural space. If trauma results in subcapsular injury and Glisson's capsule remains intact, a collection of blood between this capsule

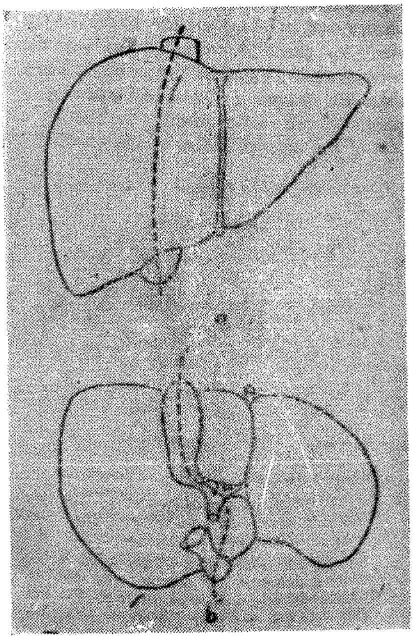

Fig. 1—The true lobation of the liver
(a) Parietal Surface; (b) Visceral Surface

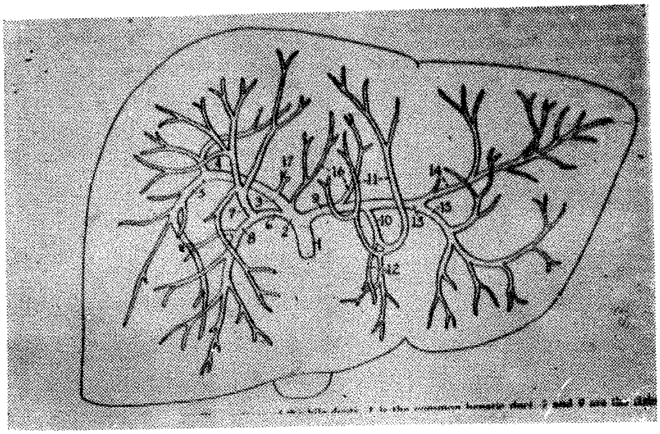

Fig. 2—The pattern of the bile ducts

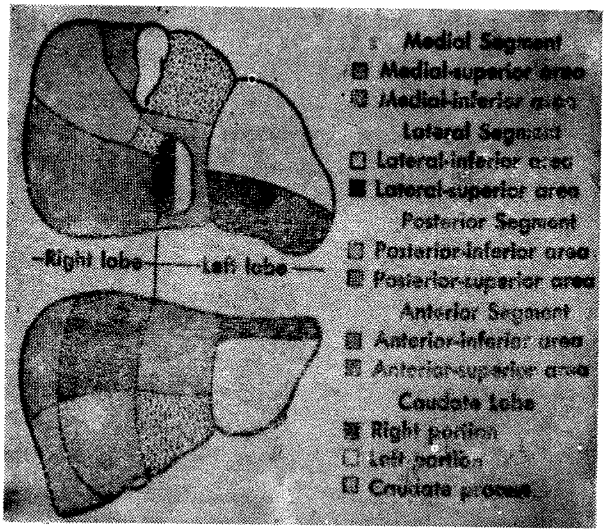

Fig. 3—Divisions of the liver, based upon the biliary drainage of the organ Upper—Visceral Surface; Lower—Parietal Surface

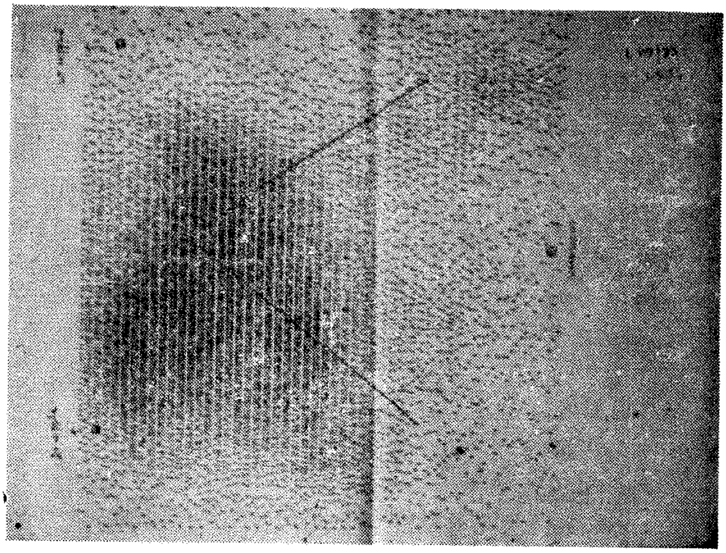

Fig. 4 (a)—Liver scans showing filling defect in Right lobe. —Anterior projection and the parenchyma is usually found on the superior surface of the organ. In this situation, the capsule is stripped from the parenchyma by the fluid which accumulates in this newly created space. Subcapsular ruptures are much rarer than wounds which interrupt the Glisson's capsule.

The third pathologic type has been termed "central rupture". This consists of interruptions of the parenchyma of the liver which lead to hematoma and then to abscess and cyst formation. Central ruptures do not bleed into the abdominal cavity initially, and, unless death occurs, complications develop slowly. By careful examination of autopsy material, it has been demonstrated that this type of lesion is not so uncommon as had been previously considered. The development of hemobilio is related to this lesion.

Another interesting complication of trauma to the parenchyma of the liver is the production of emboli of various sorts which gain entrance to the hepatic veins and are carried to the right side of the heart and lung.

Advances in the Diagnosis in the Hepatic Trauma

Penetrating wounds of the liver pose no problem in diagnosis especially when they are located in the Rt lower chest or upper abdomen. Probing or injection of suitable contrast medium followed by anterior and lateral radiographs confirm the involvement of the liver. It is blunt injuries of the liver that present a challenge in diagnosis and it is here that the recent advances have improved a pre-operative diagnostic rate of 46.3% to 83.6% (BALASEGRAM 1969, 1970).

These diagnostic aids are :—

(a) Abdominal paracentesis:—A point in each of the four quardrants of the abdomen is anaesthesised and with a 14 gauge needle or a 18 gauge spinal puncture needle, abdominal paracentesis is done in the following manner. The patient is tilted to his right side thus permitting the blood to gravitate downwards. The needle is fully introduced and withdrawn slowly, aspirating all the while. If blood is obtained, the remaining quadrants are not aspirated.

(b) Ringer's lactate infusion test :—This is a non–specific test. An immediate rise in blood pressure following the infusion of Ringer's lactate solution indicate the presence of internal haemorrhage.

(c) Peritoneoscopy :—This is helpful in the diagnosis of injuries located in the anterior or inferior surfaces of the liver. However, its role is limited, since the dangers of air embolism are ever present.

(d) Hepatic scanning :—Hepatic scanning using radio–active Indium 113 M has been helpful to exclude liver injury, in whom clinically it is thought to be present (Fig IV and Fig. V, Va, Vb, Vc). Positive sign is shown as "Cold Areas" in intrahepatic bleeding, in subcapsular haematomas the scan margin and the liver edge do not coincide.

(e) Angiography :—The use of angiography in the diagnosis of traumatic lesions in the abdomen, and of the liver in particular is a new concept (Freeark, 1969) and its full potential is yet to be realised. Splinoportography, inferior venocavography, and retrograde arteriography after percutaneous catheterisation of the femoral artery by Seldinger's techniques are the methods commonly employed for the study of liver injuries (Fig. VI). A positive diagnosis of liver injury is shown as disruption of the portal venous system with extravasation of the dye (Fig. VII). Operative hepatic or coeliac arteriography has been found to be of great assistance in the evaluation of liver injuries, in the selection of patients for massive resection, and in defining the segment and the amount of liver tissue to be resected.

(f) Specific enzyme studies :—In the presence of liver trauma there is a marked elevation of lactate dehydrogenase while the transaminases, S.G.O.T. and S.G.P.T., are raised to a lesser degree.

(g) Methylene blue test :—Methylene blue test—An enlarged liver in subcapsular haematomas can be shown on radiology and scanning, but it is necessary to exclude intralobar rupture of the liver parenchyma. In such cases extravasation of large amounts of methylene blue beneath the liver capsule following injection of the dye into the common duct is indicative of intralobar rupture. The technique is accurate and less time consuming than operative cholangiography.

The use of these diagnostic aids, and especially hepatic scanning, angiography and enzyme studies, are of significant value in excluding liver damage in patients with multiple injuries. The information obtained may not only establish the need for operative intervention but also locate the site, type and extent of the lesions. Such knowledge is invaluable in assigning priorities and in outlining a pre-operative plan.

However, it must be stressed that in spite of the use of such aids 16.4% of blunt liver injuries were discovered on exploratory laparotomy.

Treatment

Pre-operative treatment consists of urgent resuscitation with massive and rapid blood transfusions. A quick assessment of other associated injuries is carried out to decide which organ has to be attended to first. Surgical intervention, when indicated, must be undertaken as soon as possible. Central venous pressure monitoring, urinary output measurements and blood gas analyses are essential in all patients with severe injuries.

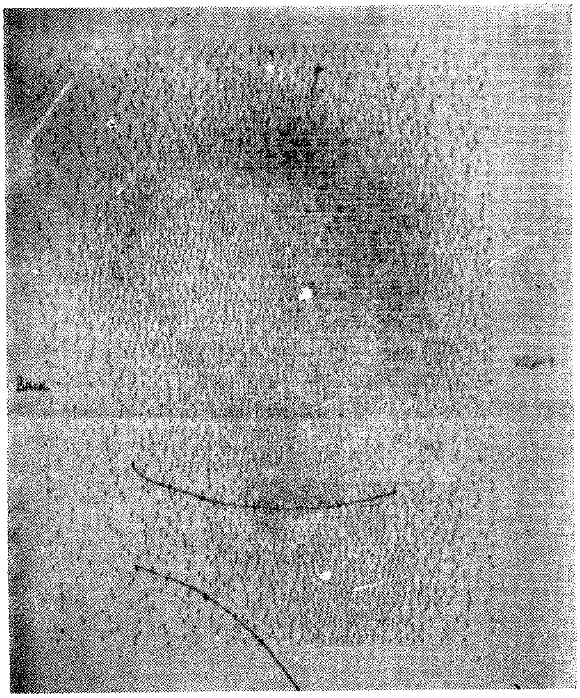

Fig. 4 (b)—Liver scans showing filling defect in Right lobe —Right lateral projection.

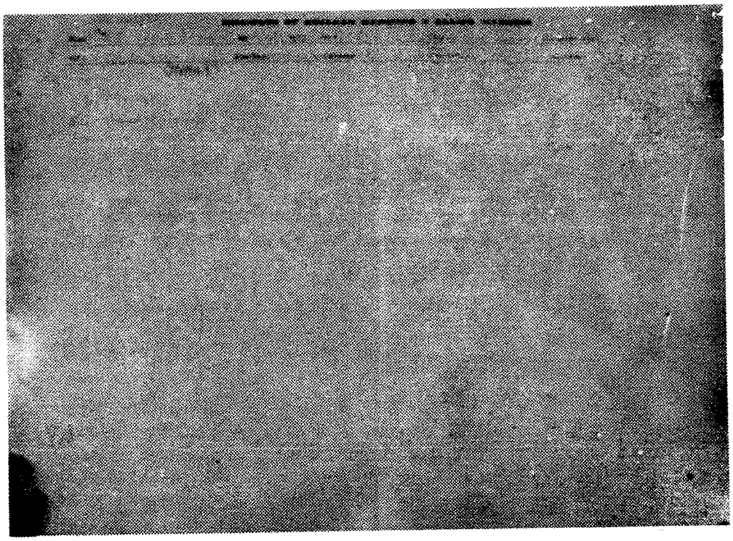

Fig. 5 (a)—Liver scans showing a huge defect in Right lobe —Right lateral projection

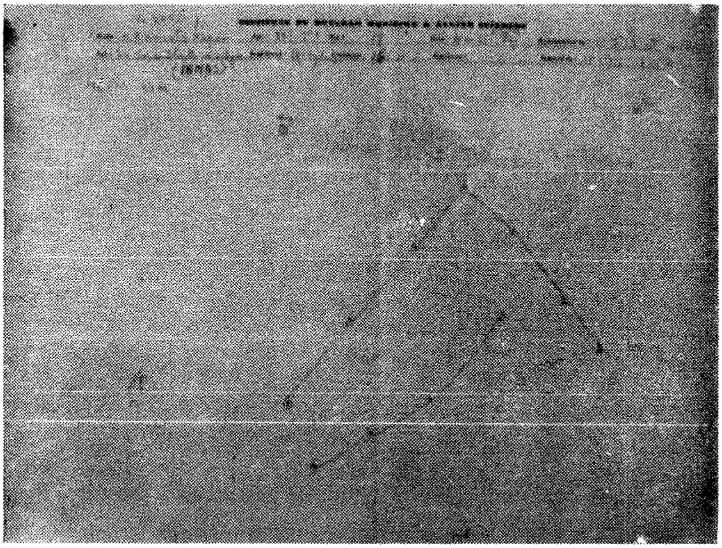

Fig. 5 (b)—Liver scans showing a huge defect in Right lobe
—Anterior projection

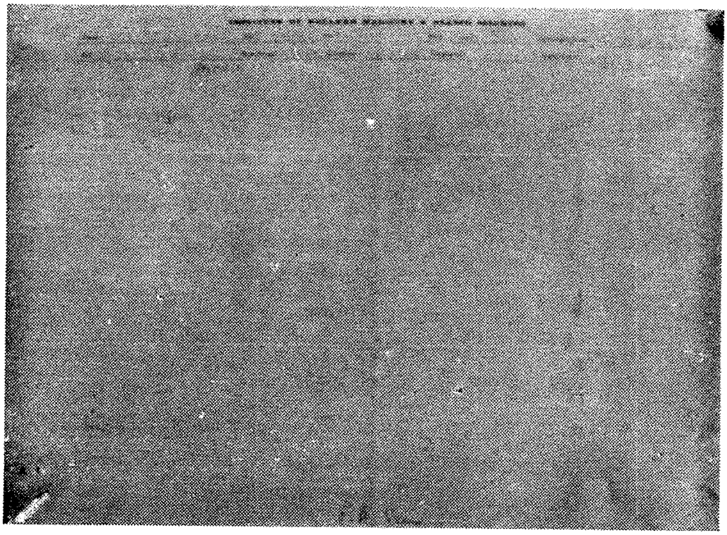

Fig. 5 (c)—Liver scans showing a huge defect in Right lobe
—Posterior projection

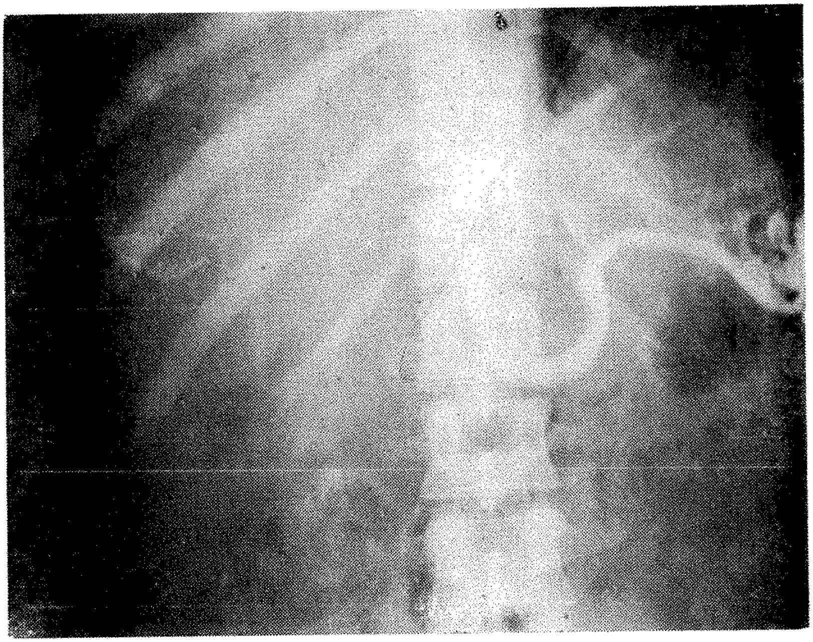

Fig. 6—Splenoportogram showing filling defect in Right lobe.

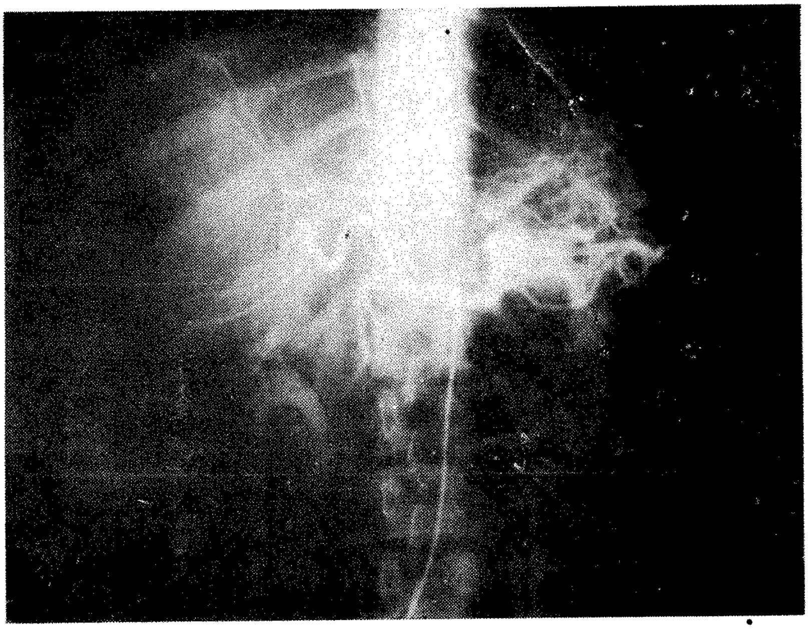

Fig. 7—Coeliac Angiography showing an avascular filling defect in Right lobe of liver.

(a) Exploration and drainage—When the injury is small namely less than 4" in length and 1/2" in depth and if active bleeding is ceased, surgical intervention is considered unnecessary, drainage of the subhepatic space/site of injury is all that is required.

(b) Suture and drainage : Suture is necessary because of active bleeding from the liver and those cases where the depth of injury is more than 1" depth.

The bleeding is controlled by several deep interrupted mattress sutures of 2/0 chromic catgut.

(c) Debridement and suture : In this Devitalised tissue is progressively removed until pink, bleeding liver tissue shows. The wound edges are then brought together in close apposition and sutured. It is imperative that the closure should be by accurate apposition namely bile duct to bile duct and vessels to vessels, to prevent haemobilia or the subsequent development of traumatic liver cysts, if the wound is deep drainage is also essential.

(d) Packing : It often fails to control bleeding and death results from haemorrhage, multiple abscesses, haemobilia and bile peritonitis, and is best avoided.

(c) Hepatic resection : The indications for resection are extensive injuries in which a major portion or a whole lobe of the liver is lacerated or when bleeding is excessive and a large portion of liver tissue is devitalised. In high velocity missile injuries, apart from the obviously damaged wound, there is a zone of liver tissue around the missile track which looks normal but which later will undergo necrosis. This deceptive appearance is the result of damage to the liver cell by vibration pressure or heat and this has to be resected.

Clinical Experiences

From Dec 71 to Dec 73 a total of 18 patients of Hepatic injury have been treated in the Army Hospital. Out of these 18 patients 16 had penetrating wounds and 2 had sustained blunt trauma to the abdomen. All patients were aged between 25 to 30 years except one who was aged 17 years. The pattern of injuries varied depending upon the nature and severity of the injuring force. Penetrating injuries were the result of gunshot wounds in battle, except in one case which was a stab wound of the right Hypochondrium. Blunt injuries were caused by vehicle accidents.

Pattern of liver injuries

Single linear lacerations	— 5
Stellate wounds	—11
Subcapsular haematoma	— 2

Surgical treatment

Exploration and drainage	— 4
Suture with drainage	—11
Debridement	— 2
Resection	— 1

Complications

Haemorrhage	—10
Multiple Abscesses	— 4
Haemobilia	— 1
Sub-Phrenic Abscesses, Secondary haemorrhage and wound infection	— 2
Generalised bleeding due to coagulation deficiencies	— 3
Serum Hepatitis	— 1
Incisional Hernia	— 2

In this series there were 4 deaths, 2 were due to secondary haemorrhage with generalised bleeding due to coagulation deficiencies. One patient died of Serum Hepatitis 4 months after successful debridement and suture. One patient died as a result of prolonged sepsis due to Subphrenic Abscess, and secondary Hepato-Renal failure. All the survivors have been followed up from one to two years and have not shown any ill effects of Hepatic trauma. 10 of the survivors developed major complications in the immediate post-operative period, which were successfully managed.

usions

1. The early diagnosis of hepatic injuries is of paramount importance.
2. Concomitant and associated injuries must be excluded but, if present, priorities in treatment listed. Such injuries carry a grave prognosis.
3. Pre-operative resuscitation of patients with hepatic injuries should be immediate, with no half-way measures.
4. If indicated, surgery should be as aggressive as the condition warrants.
5. Control of the bleeding liver, prevention of sepsis and adequate drainage are most important factors in dealing with the traumatic liver.
6. Simple linear wounds need only be sutured and drained.

7. Extensive lacerated (often macerated) or shattering injuries of the liver and subcapsular haematomas with intralobar rupture should be treated by resection of the involved segment. Debridement of the devitalised tissue is vital to prevent sepsis and subsequent secondary haemorrhage.

Acknowledgement

I wish to thank Lt Gen A K Barat, PVSM, MVC, PHS, FRCS, Chief Consultant in Surgery to the Armed Forces, for his continued support in the management of these cases. My thanks are also due to my Surgical and Medical colleagues for their help in the management of these cases.

I also wish to thank the Commandant of the Army Hospital, Delhi Cantt for permitting me to publish these cases.

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STRICTLY POSTERIOR MYOCARDIAL INFARCTION A DIAGNOSTIC DILEMMA

BY

Major V. Balasubramanian*, Major V.D. Tiwari and**

Lt Col P.K. Khanna***

It is universally recognised that myocardial infarction may involve four distinct anatomical zones of the left ventricle—the anterior, lateral, apical and diaphragmatic surfaces.[1], [2], [3] It is generally not realised, however, that the infarct may either involve or extend to the true posterior surface of the heart. This confusion is due to the diagnostic criteria of a strictly posterior myocardial infarction not being discussed even in certain reputed text books of Cardiology and use of the term posterior infarction interchangeably with a diaphragmatic wall involvement even by leading cardiologists.[4,5,6,7,8,9,10,11].

Transmural myocardial necrosis characteristically produces 'Q' waves of abnormal depth and duration in the conventional leads. These 'Q' waves are attributed to the initial part of the QRS Vector being directed away from the electrically silent myocardium. If this area is located posteriorly the initial Vector is directed anteriorly and no Q waves will be produced. The oesophageal lead will, however, show the pathognomonic 'Q' wave over the ventricualr area. The absence of the classical Q waves in the routine electrocardiogram causes strictly posterior myocardial infarction to be commonly overlooked by the unwary.

These problems of incorrect nomenclature and pitfalls in diagnosis prompted us to scan each case of suspected myocardial infarction carefully for detection of true posterior infarction.

Material and Method

All cases of acute myocardial infarction admitted in CH (SC) Pune and Army Hospital Delhi Cantt were studied in detail. A total of 12 cases were detected to have

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changes suggestive of this entity. The diagnosis was confirmed in all cases by typical clinical picture, rise in transaminase levels and serial electrocardiograms. In seven patients oesophageal electrocardiograms were obtained to delineate the changes more accurately. Palnitkar's oesophageal electrode was used in three cases and an imported electrode in four cases.

The patients ranged from 32—65 years of age with one female and eleven male patients. The diagnosis was initially missed by the attending physician on emergency duty despite the presence of typical ECG changes in three cases. Three out of the twelve died and the rest recovered uneventfully. No arrhythmias were noted in any of the cases ; right ventricular hypertrophy, right bundle branch block and Preexcitation syndrome type A were carefully excluded.

Diagnostic features

The electrocardiographic changes seen in our group of patients are summarized in Table I.

Table I

Summary of changes in routine ECG

Serial No	Parameter	Number of cases	Percentage
1.	Tall R–V1 (> 7mm)	12	100
2.	R : S>1 in V 1/V2	12	100
3.	Tall R V2 (> 10 mm)	12	100
4.	Slur on descending limb of R	6	50
5.	R > .04 Sec in V1	8	66.7
6.	Upright T in V1	12	100
7.	ST depression in V1/V2	7	58
8.	Tall 'T' in V2/V3 (> 10 mm)	9	75
9.	Associated involvement		
	(a) Anterior	4	33.3
	(b) Inferior	6	5.00
	(c) Multiple	2	16.7
10.	Arrhythmias	0	0

Oesophageal electrocardiograms were obtained in seven patients and the findings are summarized in Table 2.

Table : 2

The Oesophageal electrocardiogram in Strictly Posterior Infarction

Srl No	Parameter	Ventricular	Transitional
1.	'Q' waves	7	7
2.	QS pattern	4	4
3.	Qr pattern	3	3
4.	ST elevation	4	3
5.	T inversion	7	7

Fig No 1 and 2 depict the changes of strictly posterior infarction in routine and oesophageal electrocardiograms.

Discussion

Strictly posterior myocardial infarction is considered to be quite uncommon (12). Its true incidence is, however, very difficult to assess as the electrocardiographic diagnosis is commonly missed. This could be attributed mainly to the fact that when posterior infarction occurs as an isolated lesion, no Q waves are encountered on the routine ECG. Further, when the combination of either postero-diapharagmatic or postero-lateral infarction occurs the extent and severity of infarction may be misjudged. Finally signs of re-infarction or old healed infarction may be erroneously diagnosed as right ventricular hypertrophy. Though vector cardiogram is considered by some to be essential for recognition of strictly posterior infarction, it is now recognised that a conventional 12 leads scalar electrocardiogram is also capable of providing diagnostic clues[13]. The following criteria have been suggested for diagnosis of this condition in a routine ECG[12].

1. An R wave of 0.04 second or wider in V1 or V2.
2. R/S ratio in V1 or V2 more than one.
3. A slur or notch occurring on the descending limb of the R wave in V1.
4. Upright 'T' wave in lead V1.
5. ST segment changes in leads V1 to V3.

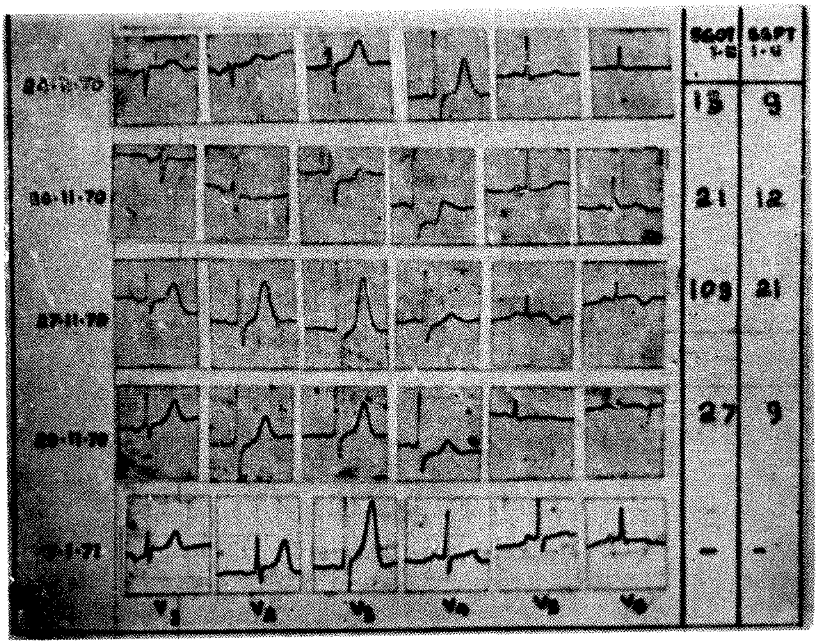

Fig. 1—Serial ECG changes in strictly posterior infarction. Note the ST depression in $V_2 - V_4$ on 26-11-70 and appearance of tall R V_1 and tall 'T' waves in V_2 and V_3 with a diagnostic rise in SGOT levels.

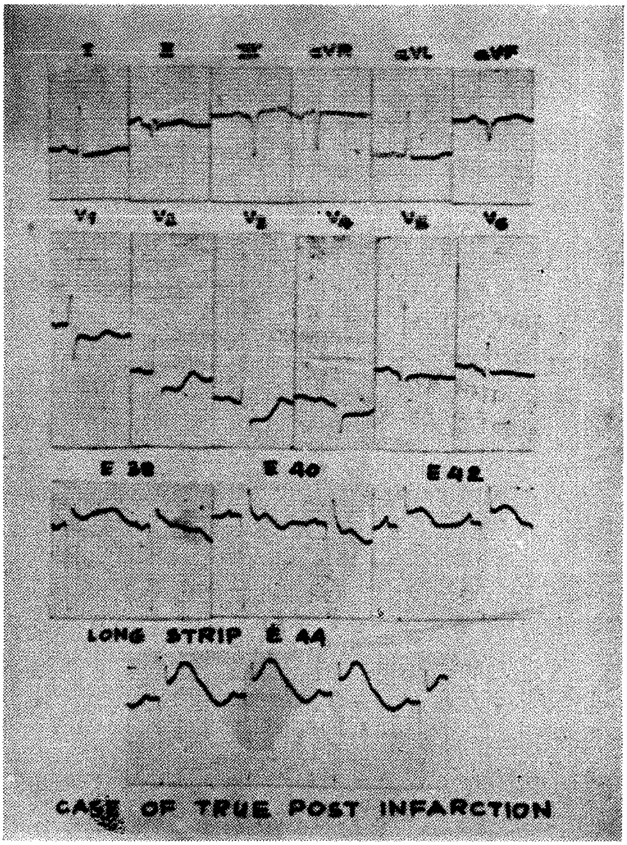

Fig. 2—Oesophageal electrocardiogram in strictly posterior infarction.

The routine ECG shows tall RV_1 and RV_2 with ST depression. The oesophageal electrocardiogram shows deep Q with ST elevation in ventricular (E44) and transitional zones (E42).

6. No pathological Q waves in the standard 12 leads, unless a diaphragmatic or lateral infarction co-exists.

7. Tall 'T' waves in V2 or V3.

Certain authorities feel that a routine scalar electrocardiogram may not be accurate enough to detect posterior infarction and advocate orthogonal ECG or oesophageal ECG. In a study of 260 cases of myocardial infarction, Erikssen et al[14] have correlated the scalar ECG findings with orthogonal ECG. They observed that "Scalar signs not commonly used may be of importance", and singled out Perloff's criteria to be as accurate as orthogonal ECG in diagnosing posterior infarcts, the level of accuracy being 88%.

Classically the ST segment is elevated over an area of acute infarction signifying an "injury pattern". In contrast an ischaemic pattern is indicated by the presence of ST depression. The changes in posterior infarction being a mirror image of an anterior one, ST depression in V1 or V2 in such cases signifies myocardial injury and not ischaemia. Persistence of this downward shift for a long time indicates a ventricular aneurysm posteriorly as contrasted to a persistent ST elevation seen in anterior or diaphragmatic aneurysm.

It should be realised that a dominant R in V1 may be encountered in four other conditions also. A normal child may show a dominant R-V1 but the clinical picture cannot be confused with a posterior infarction. The other situations in which a tall R-V1 or V2 are encountered are Wolff-Parkinson White syndrome with anteriorly directed delta waves, right bundle branch block and right ventricular hypertrophy. Electrocardiographically the first two are easily recognised. It should be noted, however, that a right bundle branch block co-existing with a posterior myocardial infarction is the only situation where the former may mask or confuse the diagnosis of myocardial infarction. In case of right ventricular hypertrophy upright 'T' waves in V1 and left axis deviation are uncommon findings and should arouse the suspicion of posterior myocardial infarction. Diligent clinical examination and, serial ECG and enzyme studies are of great help as additional diagnostic aids.

Some workers feel that a diagnosis of localised posterior myocardial infarction cannot be made unless some portion of the inferior or lateral wall is also involved[14]. Evidence of the latter sites in addition to the criteria already enumerated should lend further support to the diagnosis.

In our experience oesophageal electrocardiogram has proved to be very valuable in the diagnosis of the entity. The presence of pathological Q waves at the ventricular level and ST elevation with T inversion at ventricular or transitional level is diagnostic.

It is recommended that oesophageal ECG should be performed routinely in such cases as it enables the physician to explore the posterior aspect of the heart thoroughly and systematically.

SUMMARY

Myocardial infarction involving the posterior wall of the left ventricle is relatively uncommon and is usually missed on routine electrocardiogram due to the absence of pathognomonic Q waves. Twelve cases of this variant of myocardial infarction are presented and the diagnostic criteria on routine scalar Electrocardiogram elucidated. The value of oesophageal electrocardiography in diagnosis of these cases is stressed. Attention is drawn to the pit-falls in diagnosis and a plea is made for a wider awareness and correct nomenclature of this entity.

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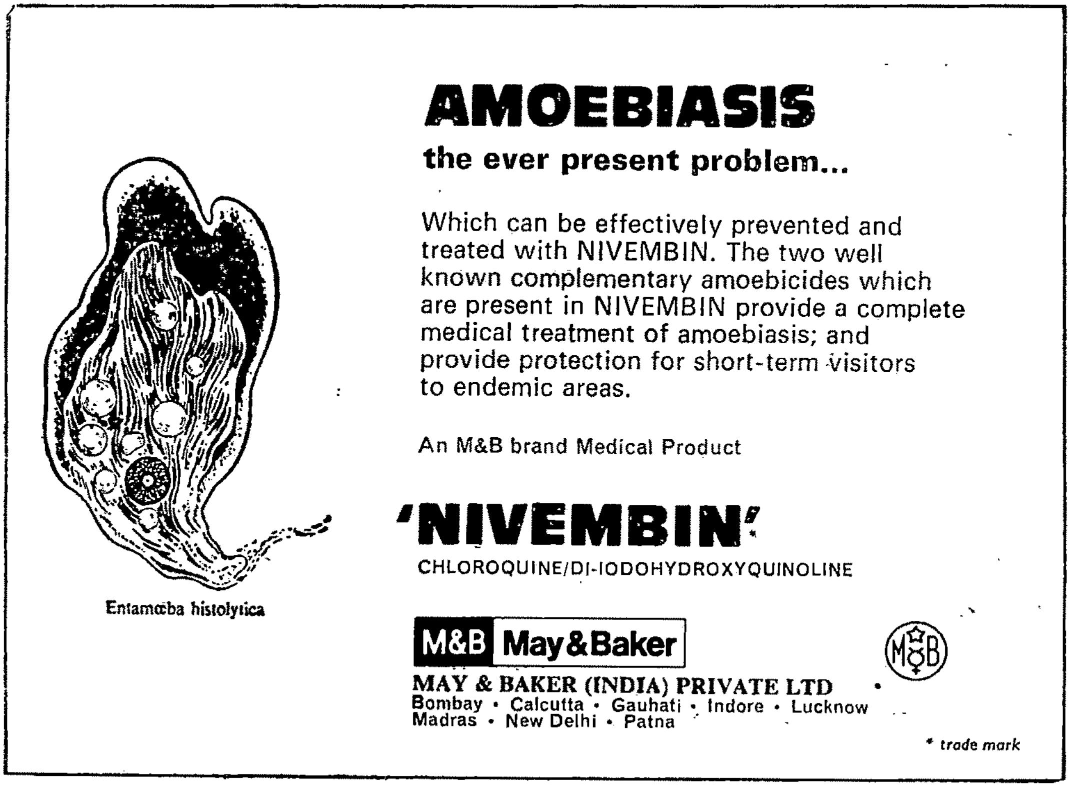

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ACUTE RESPIRATORY FAILURE

BY

Lt Col K.L. Sobti AMC*

INTRODUCTION

Acute respiratory failure is a medical emergency which may occur in a variety of clinical settings (7, 20). The syndrome of acute respiratory failure and its management remain somewhat obscure in the minds of most physicians due to considerable controversy about the management in available literature.

In recent years an organised approach to the management of acute respiratory failure has emerged as a significant contribution. Acute respiratory care units have been established at various hospitals providing sophisticated means of managing the difficult malady (4, 24).

The rehabilitation potential for patients with chronic respiratory diseases who suffer respiratory failure has only been recently emphasised. It is generally agreed, however, that acute respiratory failure is not to be equated with irreversible pulmonary damage and that once patients recover from the precipitating events which lead to respiratory failure a full recovery is possible.

DEFINITION

It is present when arterial oxygen tension (P02) is less than 50 mm Hg (normal is 85-100 mm Hg) with or without arterial carbon dioxide tension (PCO2) more than 50 mm Hg (normal is 38-42 mm Hg) and accompanying pH of less than 7.30 (normal is 7.35-7.45) (20, 21).

AETIOLOGY

The aetiological factors producing blood gases abnormalities include defects in ventilation, in diffusion and in ventilation/profusion ratio (VA/Q) or intrapulmonary shunting (venous admixture). Various combinations of these factors often occur in the same patient. The disease states and the associated pathophysiological classification producing respiratory failure are outlined below (20, 21).

---

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Ventilation Defects

(a) Chronic airway obstruction —emphysema, chronic bronchitis, asthma.

(b) Restrictive defects:

  (i) Decreased lung expansion—interstitial fibrosis, pleural effusion, pneumothorax, fibrothorax.

  (ii) Limited thorax expansion—kyphoscoliosis, multiple rib fractures, thoracic surgery, spinal arthritis.

  (iii) Decreased diaphragmatic movements—abdominal surgery, ascites, peritonites, severe obesity.

(c) Neuromuscular defects—polio, Guillain Barre syndrome, multiple sclerosis, mysthenia gravis, botulism, tetanus, brain or spinal injuries, drugs or toxic agents e.g., curare, acetylcholine esterase inhibitors, colistin, kanamycin, streptomycin, neomycin etc.

(d) Respiratory centre damage or depression—narcotics, barbiturates, tranquillisers, anaesthetics, cerebral infarction or trauma, high flow uncontrolled oxygen therapy.

Impaired Diffusion and Gas Exchange

(a) Pulmonary fibrosis—sarcoidosis, Hamman Rich Syndrome, pneumoconiosis.

(b) Pulmonary oedema.

(c) Obliterative pulmonary vascular disease.

(d) Anatomic loss of functioning lung tissue—pneumonectomy, tumor.

Ventilation/Perfusion Abnormalities and Venous Admixture

Emphysema, chronic bronchitis, bronchiolitis, atelectasis, pneumonia, thromboembolism, post-perfusion syndrome and congestive atelectasis.

CLINICAL DIAGNOSIS

The diagnosis of respiratory failure may be obvious or subtle. The clinical features depend on the underlying disease and the precipitating causes. Many signs and symptoms have been attributed to the physiological effects of hypercapnoea, hypoxia and acidaemia (7, 10, 20, 21). The clinical manifestations of arterial hypoxaemia and hypercapnoea are as under.

Hypoxaemia	**Hypercapnoea**
Restlessness—impaired motor function and Judgement.	Headache
	Dizziness
Confusion, delirium	Confusion
Unconsciousness	Unconsciousness
Hypotension	Twitching
Techycardia	Miosis, engorged fundal veins, papilloedema
Central cyanosis	Hypotension
Warm extremities	Sweating

These are signs of altered circulatory or CNS function. This explains why so many cases of respiratory failure are misdiagnosed and confused with congestive cardiac failure, cerebral vascular accidents and toxic pneumonia. Any combination of these signs may be present in one patient.

Cyanosis is an unreliable clinical sign. It has been stressed that significant arterial O_2 unsaturation can exist without cyanosis (7, 21, 23).

With moderate acidaemia and hypercapnoea the patient may have no complaints or may have manifest headache, somnolence, mental confusion, tachycardia, increased sweating, warm extremities and/or twitching. During marked hypercapnoea and acidaemia, coma, depressed respiration, circulatory hyporeflexia, flaccid paralysis, tremor and convulsions may occur and finally if untreated death supervenes. The level of consciousness is more related to lowered pH than to actual CO_2 tension (8, 14, 20).

LABORATORY DIAGNOSIS

The following investigations are usually adequate :—

(a) Arterial blood pH, PO2, and PCOs

(b) Serum electrolytes

(c) Urine electrolytes

(d) Other investigations such as, haemoglobin, haematocrit etc.

MANAGEMENT.

The basic principles of management of acute respiratory failure are to treat the potentially reversible features, while providing external support to correct hypercapnoea and hypoxaemia. Before planning the therapeutic approach, the nature and severity of the underlying disease if any, precipitating factors and the reversible aspects must be kept in view. The actual management may be divided into three parts :

(a) Immediate and emergency care

(b) Definitive Care

(c) Early rehabilitation.

Immediate and Emergency Care

It includes the (i) the management of airway, (ii) management of hypoxaemia/adequate CO2 removal, and (iii) control of hydrogen ion concentration.

Management of airway.—The first responsibility of the treating physician is to secure an adequate airway so that efforts to provide oxygen or measures to improve ventilation can succeed. Various methods are available (7, 17, 20) :

(a) **Unconscious patient**—The frequent problem is upper airway obstruction in these cases. When ventilation is unimpaired and adequate cough and gag reflexes are present, the use of oropharyngeal or nasopharyngeal airway may be adequate. When gag reflexes are absent or swallowing ability is impaired, an airway with inflatable tracheal baltoon is needed. If secretions are a problem tracheal intubation or tracheostomy are advisable.

(b) **Patients with increased secretions**—When conservative measures such as steam or mist inhalations, physical therapy, nasopharyngeal suction, IPPB (intermittent positive pressure breathing) and bronchoscopy have failed, tracheostomy is indicated. An endotracheal tube is generally unsatisfactory.

(c) **Patients needing prolonged assisted ventilation**—When respiratory assistance is for less than 48 hours orotracheal or nasotracheal ventilation is necessary. For ventilatory assistance beyond 48 hours a treacheostomy will be more satisfactory.

(d) **Patients with upper airway obstruction**—Tracheostomy is essential.

Management of Hypoxaemia/adequate CO2 removal.—Hypoxaemia may occur with or without hypercapnoea. The toxic effects of hypoxaemia and tissue hypoxia are numerous. Hypoxaemia must be corrected. This should be done with low flow carefully monitored oxygen therapy (1-3 litre/min). Arterial oxygen saturation of 100% should not be attempted for. Such flow of oxygen corrects hypoxaemia in the majority of cases with chronic airway obstruction without the hazards of CO2 narcosis. Progressive PCO2 rise on low flow controlled oxygen therapy is rarely encountered. When such patients are encountered, assisted or controlled ventilation is essential. Deprivation of oxygen therapy in a patient with significant hypoxaemia (PO2 less than 50 mm Hg) due to fear of CO2 intoxication may cause death due to hypoxaemia (7, 12).

The use of intermittent oxygen as a method of eliminating the possible rise in PCO2 is also extremely hazardous. The sudden cessation of oxygen inhalation may produce more severe hypoxaemia and catastrophic results may ensue. The following are the methods adopted for giving oxygen (15) :—

(a) **Nasal Catheter**—It is an effective method of giving oxygen but the patient is not comfortable.

(b) **Venturi or Edinburgh masks**—Oxygen can be effectively administered as 24%, 28% and 35% concentration. The masks are uncomfortable, expensive and have to be removed during eating.

(c) **Reservoir**—Reservoir face masks are of value when high O2 concentrations are used such as in shock states or severe interstitial disease or marked diffusion impairment.

(d) **Two-pronged nasal cannula**—This is the simplest and most comfortable. It should be remembered that the patient should not receive prolonged oxygen concentration higher than 33 per cent.

Ventilatory support.—The need for ventilatory support may be an emergency situation. Mouth to mouth respiration or the hand type self inflating Ambu Bag is all that is needed. Once control of the airway is secured the self-inflating bag is all that is required till a more permanent type of ventilator is set up (7, 20).

Mechanical ventilators—A variety of machines are available for the ventilatory support. In principle the function of all the machines is to aid the work of breathing. They force air into the lungs in an attempt to give the patient adequate tidal volume. Commonly the following types of machines are used :—

(a) **Pressure cycled**—The pressure cycled devices manufactured by BIRD and BENNETT are commonly used. They are triggered by the patient's inspiratory effort and are automatically set to control respiratory rate. Air enters the lungs until a preset cycling pressure is achieved whereupon the valve closes. The advantages are simplicity and familiarity by medical personnel. The disadvantages are interference with flow, low pressure capability and very poor control of inspired oxygen concentration.

(b) **Time cycled devices**—The machines are manufactured by AIRSHIELDS and ENGSTROM. They allow adjustment of the time for inspiration and expiration and thus variable ratios are possible.

(c) **Volume cycled devices**—The deliver a metered tidal volume of air upto the machine's pressure capabilities. In common use are BENNETT-MI which has a pressure capability of 80 cm and OHIO 560. These devices provide the same tidal volume breath by breath despite changing airway resistance. They have the additional advantage of providing adequate tidal volume at high peak airway pressure (40-70 cm of water) but have the disadvantages of having fixed inspiration/expiration ratios and cannot be patient triggered.

In brief, sufficient ventilation is provided to bring the PCO2 towards normal at the rate of 5-10 mm Hg per hour. This can be more rapid when the PCO2 is more than 80. When the patient is profoundly acidaemic, much more aggressive lowering of the PCO2 is indicated than when some degree of bicarbonate retention has already occurred spontaneously or by exogenous HCO_3 administration. If the patient is only mildly acidaemic (pH of 7.25-7.30), the PCO2 should be lowered slowly.

Discontinuance of ventilatory assistance—After the precipitating causes of respiratory failure have been treated, most patients can rapidly attain adequate ventilation without assistance which can be measured with Wright Respirometer. The aim is to maintain adequate oxygenation, while gradually lowering the PCO2. Rapid change in PCO2 should be avoided as it can precipitate vascular collapse, convulsions and ventricular fibrillation. The level of PCO2 prior to acute respiratory failure should be considered. Many patients with chronic airway obstructive disease have become accustomed to living with a PCO2 of 60-70 mm Hg. This level should be left undisturbed.

Respiratory stimulants—The value of respiratory stimulants is controversial and they are hardly required in routine cases. Campbell, however, still recommends them in moribund cases for a period of 24 hours (7).

Control of Hydrogen Ion Concentration—The level of consciousness is more related to blood pH than to PCO2. Alterations in sensorium are usually marked below a pH of 7.2. Administration of $NaHCO_3$ and oxygen, in order to keep pH above 7.2, are frequently needed. The following dosage schedule is advocated (2) :—

(a) If pH is 7.10 to 7.15—88 mEq immediately.

(b) If pH less than 7.10—132-176 mEq is given.

The pH is corrected to a level greater than 7.25 immediately.

Definitive Care

This includes the management of precipitating and reversible factors.

Infection—It is the commonest precipitating factor in acute respiratory failure. The aetiological agents have been found viral in some studies. Sputum cultures and appropriate antibiotics are administered. For an acute infection tetracycline 2 G per day orally for an adult is the initial drug of choice. Ampicillin is also effective. Erythromycin and cephalosporin are indicated in patients with penicillin hypersensitivity. It should be remembered that treatment of infection does not end with institution of antibiotics. Methods designed to improve bronchial hygiene and relieve reversible airway obstruction are equally important.

Bronchospasm—The following drugs are used :—

(a) Adrenaline 1/2 to 1 ml (1/1000 solution) subcutaneously.

(b) Aminophyline is a potent bronchodilator. This is usually given as 500 mgm in 500 ml of dextrose I.V. in 4-6 hours upto a maximum of 2 G per day.

(c) Inhalation of epinephrine 2.25 % or isoproterenol 0.5% or Salbutamol by IPPB nebulization (diluted 1 in 2 or more in distilled water) is effective.

(d) Salbutamol (Ventolin) orally also is an effective bronchodilator.

(e) Corticosteroids should be used when bronchiolitis is present. Prolonged use of steroids in chronic airway obstruction is hazardous and rarely advisable.

Retained secretions—Retained secretions are a major problem in patients with chronic airway obstructive disease. In chronic bronchitis secretions are extremely tenacious, difficult to cough out and may seriously interfere with adequate ventilation. Sterile saline may be introduced into the upper airway via a small transtracheal catheter inserted below the cricoid cartilage or directly through a tracheostomy tube. If the patient's cough is ineffective in removing secretions mechanical suction may be used. Drying agents such as, antihistamines and atropine should be avoided. Acetyl cystein (mucomyst) diluted with equal quantity of normal saline is fairly well tolerated and is quite effective (20, 21, 24).

Congestive cardiac failure—Chronic cor pulmonale is a well kuown complication of chronic lung disease. The occurrence of repeated episodes of congestive cardiac failure in patients with chronic bronchitis, chronic hypoxia and polycythaemia is common. It is now clear that both hypoxia and increased hydrogen ion concentration cause pulmonary vasoconstriction and a rise in pulmonary pressure. The possibility of arrythmias should be anticipated in patients with hypoxaemia or acidaemia. Hypoxaemia must be corrected before digitalisation. These measures will often provide increase in cardiac output and diuresis. The cardiac glycosides improve cardiac output in right heart failure. Diuretics such as, thiazides, trimeterene, frusemide, ethacrynic acid and spironolactone may also be used (3, 20, 21, 22).

Correction of electrolyte abnormalities—Any pattern of electrolyte abnormality can occur. Most common is severe potassium depletion due to renal potassium loss during the development of acute respiratory failure or existing potassium deficit from use of thiazides or corticosteroids. Potassium replacement should begin immediately if initial potassium level is not elevated. It should be given as chloride salt.

Secondary polycythaemia—Removal of 500 ml of blood by venesection is indicated in a patient with haematocrit in excess of 60% but with normal cardiac output. This may be repeated every second day.

Poisoning—Specific treatment for poisoning should be instituted.

Sedation—No sedatives should be given.

Early Rehabilitation

Patients with surgical/neurological disorders and poisoning can be expected to recover as soon as the exciting cause is controlled. They should return to their previous level of activity. Patients with severe chronic lung disease require rehabilitative measures to improve function and utilise ventilatory capacity more efficiently.

Breathing retraining utilising abdominal/diaphragmatic breathing, slow breathing and exhalation against pursed lips along with graded ambulation with the use of supplemental oxygen and occasionally with mechanical assistance is begun in Intensive Care Unit. These measures plus bronchial hygiene and patient education are the principles of successful pulmonary rehabilitation and should be continued after the patient is discharged from the Unit (20, 21).

The Respiratory Care Unit—The complexities of managing patients with severe respiratory failure are best met by team approach to management. The problem involving airways care, inhalation therapy and use of variety of therapeutic agents require trained personnel to ensure the successful outcome in a given case (4, 5, 23).

The following scheme for a Respiratory Care Unit to provide all aspects of necessary medical, surgical, physical medicine and nursing care is suggested (Fig. 1).

Fig. **Scheme for Acute Respiratory Care Unit**

	Unit Director		
Surgeon	Internal Medicine Specialist		Pulmonary Physiologist
Anaesthetist	Physical Therapist	Inhalation Therapist	Laboratory Technician

SUMMARY

The aetiology, diagnosis and management of acute respiratory failure have been elucidated.

It should be realised that acute respiratory failure may occur in a variety of clinical situations. While clinical impressions are helpful, blood gas analysis is essential in arriving at an accurate diagnosis. Immediate care must include maintenance of clear airway, adequate oxygenation, control of hydrogen ion concentration and of the ventilatory assistance. In addition, specific treatment should be directed towards infection, retained secretions, bronchospasm, congestive heart failure and eventual patient rehabilitation. Acute respiratory failure does not occur solely in patients with irreversible pulmonary damage. In other words long term improvement after thorough treatment of respiratory failure may restore patients to highly useful lives.

The team approach utilising a respiratory care unit is most likely to provide a successful outcome in the management of all varieties of respiratory failure. The suggested organisation is described.

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ANAESTHESIA IN WAR AND PEACE—RECENT DEVELOPMENTS

BY

Col N.C. Das, Vr. C. and Lt Col J.K. Chandnani

INTRODUCTION

The Armed Forces in India are deployed on very long borders with different climatic and logistic problems in the different sectors. Our Surgical teams have to provide medical aid to the fighting Jawans in such arduous circumstances. We review from time to time the role and capabilities of our surgical teams. We try to fill the deficits in the techniques and equipments of anaesthetic practice that come to light in the changing and continuing perspective of forward field surgery with the experiences gained during the successive wars. Our duty towards the fighting soldier does not end in the field. The treatment in our field and base hospitals is a continuing process. In the present golden age of scientific anaesthesiology the surgical horizon in our base hospitals has extended without limit. We have reduced the mortality rate of wounded soldiers from 5.6% in 1965 to 3.2% in 1971.

The figures are comparable with those of Vietnam War where Americans possessed more sophisticated equipment and speedier methods of evacuation. All this has been possible due to improved methods of active resuscitation with better understanding of pathophysiology of shock and battle wounds where an anaesthesiologist plays a key role.

ANAESTHESIA FOR FORWARD FIELD SURGERY

(a) Resuscitation
(b) Techniques and equipments
(c) Agents.

Resuscitation

Thanks to the changed concept of pathophysiology of hypovolaemic shock, the treatment of the same has become easier in forward areas. It is indeed seen that the circulatory volume is more important than the red cell mass. As isotonic crystalloid fluid similar to the functional extracellular fluid is essential to make up the blood volume. Ringer lactate solution is good, for it is isotonic with the functional extracellular fluid which is in dynamic equilibrium with the capacitance of circulatory volume. Ringer lactate comes in handy disposable packs of ½ and 1 litre with a disposable giving set. The pack can be pushed under the patient's thigh for required pressure, only drip chamber remains vertical.

Whenever blood transfusion is necessary we use two packs of Ringer lactate solution (1 litre) to one unit of blood (½ lit) with lowest permissible limit of pack cell volume as 30%. Kidney perfusion is checked by urinary output (½ to 1 ml/minute or 1 ml/kg body weight/hr) and the filling of the right side of heart by c.v.p. when we are comparatively static.

The role of colloids like Dextravan 70 is under assessment at our Army Hospital.

Equipments and Techniques

The anaesthesiologist in Army has to be well versed with the practice of anaesthesia for each type of surgery which can possibly be carried out during war and the training in Armed Forces is based on these principles. During 1948 J & K operation the anaesthesiologist had to be proficient in use of Rag and bottle, and Oxford Vaporizer of 1939-45 war. During their assignment in Korea, Oxford Vaporizer was the sheet anchor of our paratrooper anaesthetists. This allowed use of Ether only. Portable Boyles apparatus at present authorized on the equipment scales of our field medical units allows us to anaesthetise a patient with inhalational anaesthetic agents like nitrous oxide, diethylether, trichloroethylene, halothane and in extreme situations even chloroform in any combination desired by the anaesthetist. This is ideal for semi static hospitals. But in mobile combats there is logistic distress in maintaining uninterrupted supply of anaesthetic gas cylinders and there is susceptibility of this apparatus to failure by wear and tear and traumatic transportation. We have therefore to fall back upon the equipment which is simple and sturdy and not cylinder dependent. The EMO/PDV anaesthetic outfit incorporating Oxford Bellows satisfies most of the requirements of an anaesthetic apparatus for use in difficult and disaster situations. It works on draw over open circuit system. We have replaced bellows (OIB) by self-inflating type of rubber bag (Das 1972). This is coupled to the indigenously made valve, for use in spontaneous as well as controlled respiration, which is connected to the outlet of vaporizer.

Agents

Ether is the most readily available, cheapest and safest agent indigenously produced in our country. Its use in EMO/PDV anaesthetic outfit bridges a wide gap between the inadequacy and unacceptability of open mask technique and sophisication of modern Boyles apparatus. But ether is flammable and Bhokharis used as heating devices and Petromaxes used for lighting purpose preclude its use. Moreover ether boils away on any day during summer, in the deserts of Rajasthan. We therefore started our work on non-flammable agents in 1961 as an AFMRC Project. Trichloroethylene and methoxyflurane can be used in EMO/PDV outfit, whereas an accurately calibrated vaporizer is needed for Halothane and chloroform. Methoxyflurane has fallen in disrepute leaving us with Trichloroethylene only for use in EMO/PDV. From our studies between 1961-72 Halothane has proved its place in our armamentorium when used with accurate vaporizer. But when used on its own the cost would be prohibitive and it is not a good analgesic. Trichloroethylene as single agent is not a good anaesthetic and may cause tachypnoea. We are therefore concentrating on the combination of Trichloroethylene and Halothane used in the vaporizers in series. OMV (Oxford Miniature Vaporizer) has been used for our work because it is small and would decrease the weight of equipment for forward field surgery. Preliminary work has shown encouraging results and we are taking up the trials of this combination as an AFMRC Project for its evaluation before recommending its use for all field medical units. An indigenously made small vaporizer replacing OMV with an indigenously made valve connected to it and the self inflating bag already introduced by us will cure of all ills of dependance on Foreign equipments for forward surgery.

We have not limited ourselves to inhalational anaesthetic agents for our work in forward areas. Regional analgesia, Intravenous anaesthesia, Dissociate anaesthesia and Electro anaesthesia have evinced our interest. Regional analgesia like 'brachial plexus block' is used in very small number of cases because of limited sterilisation facilities and temperamental unsuitability of patient in forward area. Intravenous anaesthesia using thiopentone sodium still continues to hold the field. Its advantages in surgery of war outweigh all the hypothetical and real disadvantages for bulk of skeletomuscular injuries encountered there. Methohexitone and Propanidid are serious contender for replacing thiopentone. The comparative studies with these drugs are being carried in patients undergoing Electroconvulsive therapy to elucidate the superiority of one over another. Introduction of Ketamine hydrochloride has brought Dissociate anaesthesia in prominence. It has proved to be of value as a sole anaesthetic agent for diagnostic and short surgical procedures. Lot of stress has been laid on the use of Ketalar for dissociate anaesthesia for war wounded (Boulton 1973). We are working on this drug for its feasibility of availability in bulk, its economic aspect and its utility for our wounded i.e. its place for traumatic surgery. No work in India has been carried out for the role of Ketamine for traumatic surgery except in our hospitals. The pioneer work in India in Electroanaesthesia and Electro-sleep has been done by us. The potentialities are fascinating specially in poor risk cases and in event of mass casualties during atomic warfare. The disadvantage of muscle hypertonicity, excessive salivation and sympathetic over activity as manifested by tachycardia and hypertension continue to be bothersome. Search is on for a current pattern which will obviate these and make soft induction and maintenance possible. Developments in this field are worth watching for with interest.

ANAESTHESIA IN HOSPITALS (BASE)

The balanced anaesthesia consisting of tetrad of hypnosis, analgesia, muscle relaxation and adequate ceflex suppression is practiced routinely by all anaesthetists in our base hospitals. Prolonged apnoea in certain cases after balanced anaesthesia causes a great problem. The measurement of Chronaxie as described by us gives an index of muscle reactivity and the effect of muscle relaxants on neuromuscular junction. This is an objective method. Recording of Chronaxie has further been used as a means of assessment in cases of Myasthenia gravis—pre, inter and post operatively.

The major physiological upheavals implicit in surgical access to vital areas such as brain, heart, lungs and great vassels can be mitigated by expert anaesthesiologists and still remain within safe limits of physiological tolerance. To achieve this, special techniques like hypothermia and hypotension are made use of with balanced anaesthesia. Use of induced hypothermia has crystallised for repairative cardiac surgery, neurosurgery, vascular-surgery and to combat the effects of cerebral injury. The controlled hypotension is indicated in cases where bleeding is difficult to control and obscures the operating field.

Cardiac surgery

The dramatic progress in cardiac surgery and opening of Cardio-Thoracic centres in Armed Forces have evinced special interest. As the range of cardiac diseases amenable to surgical correction enlarges, accurate pre-operative assessment of patients assumes great importance. This is reflected in the increase in cardiological investigations for many of which aid of anaesthetist is sought. Closed heart surgery has been a comparatively safe and routine procedure at our cardiac centres. Open heart surgery has enabled us to correct the cardiac defects leisurely. The cardiopulmonary bypass provides all the facilities. Anaesthesiologist plays a big role in maintaining biochemical values within normal limits and correcting any changes during cardiopulmonary bypass, by administering suitable drugs, electrolytes, gas and anaesthetics. He holds the thread of life when heart has been intentionally stopped. The essential monitoring which guides the anaesthesiologist during surgery consists of blood pressure, venous pressure, ECG, a-vO2 difference, pH, bicarbonate, pCO2, packed cell volume, temperature and urine flow.

Intensive Cafe Units

The patients who undergo major surgical procedures like open cardiac surgery and acutely ill cases need intensive care. The concept of progressive patient care has therefore been accepted. Armed Forces were the first to conceive this idea in India and put it in practice by setting up Intensive care unit in Army Hospital, Delhi Cantt. Here all the acutely ill patients are grouped together in one place to accomplish economy and maximum utilization of trained personnel and expensive equipments. We have further elaborated our Intensive care units by having Coronary care units. The anaesthesiologist by virtue of his special training as cardiopulmonary physiologist, in the most suitable specialist to look after the acutely ill patients needing intensive care. He is administratively also most acceptable. With better understanding of physiology of artificial ventilation, the anaesthesiologist today shoulders the responsibility of looking after cases of tetanus, status epilepticus, myasthenia gravis, repiratory paralysis, barbiturate poisoning and all cases of coma. The concept of "Intensivist" is fast evolving a suitable anaesthesiologist taking on to become one, after at least 5 years standing as clinical anaesthesiologist.

PROSPECT

It is clear from the brief resume that we have made tremendous strides in the field of anaesthesiology. This has enabled our surgical colleagues to produce results comparable to the best institutions. Our endeavour is to keep abreast of latest developments and better the standards of anaesthetic service in Armed Forces. In this direction it will be in the best interests of service to have more research workers and specially qualified anaesthesiologists for traumatics surgery, open heart surgery, neurosurgery and paediatric surgery. A cadre of "Intensivists" should be thought of in the changing perspectives.

MINE BLAST INJURIES AND THEIR MANAGEMENT IN FORWARD AREAS

BY

Major T. K. Roy Chowdhury, AMC*

INTRODUCTION

Analysis of various types of casualties managed in 319 Field Hospital shows that a considerable percentage of mine blast cases occur among battle and non battle casualties. It is presumed that in event of any hostility in future in this theatre, there is every possibility of receiving same type of casualties. Such cases do happen even during peace time, and considerable number of army personnel and local civilians have been injured in the past. Number of ponies meant for carrying ration and ammunition have also been killed due to stray mines.

It will not be out of place if a brief mention is made about the common types of anti-personnel mines. Because of the mountainous terrain in this area, only anti-personnel miner are biggest deterrent for the advancing enemy. Mineblast almost invariably causes severe degree of injuries of ankle, both ankle and foot, occasionally lower half of leg. In certain cases splinters cause instantaneous death, or soft tissue injuries of thigh, buttock and other parts of the body. The poisonous smoke after a blast can damage the eyes permanently. The casualties caused due to minefields may not be very high, but its very presence demoralises the advancing troops.

Types of mines :—

(a) M-16 (Fragmentation Mines) : This mine is of US Origin. It is a very deadly mine. It can actuate with a load of 8 to 20 lbs, or a pull to its tripwise equal to 3 to 8 lbs, Its killing area is 35 yds all round. It contains 1 lb explosive.

(b) NMM-14 (Blast type) : This mine is of UK Origin. It has got about 1 oz of explosive in a plastic container. It can actuate with a direct load of 20 to 35 lbs. The effect of this mine is not to kill but to make the enemy a liability on the troops by grossly damaging or even blowing off the leg. This mine is more demoralising and achieves better result. Although M-16 is more dangerous and its killing area is more, NMM-14 is commonly used.

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Aim

The aim of this study is to bring out the lessons learnt while tackling mineblast cases with their inherent evacuation problems from forward defended localities, and the irreparable damages to the injured limbs with infection before the cases reach the surgical team for doing primary surgery.

Materials and methods

Fifty mineblast cases have been tackled in 319 Field Hospital between Nov 1971 and May 1973; this figure does not include the number of casualties who expired immediately after sustaining injuries from M-16. An effort has been made to have a detailed analysis of mineblast cases. This analysis includes the side and portions of limbs involved, with or without associated injuries, resuscitative measures, types of amputations and results, and the follow up of a few cases with prosthesis. Because of the difficult mountainous terrain which also remains snowbound for 6 to 7 months in a year, the evacuation time of casualties from forward defended localities is unduly prolonged in times of war as well as in peace time.

Table 1 : Sites of Injury

Ser No	Parts of limbs involved	No of cases	Sides involved	Percentage
1	Fore Foot	5	Rt Side –3 Lt Side –2	10%
2	Complete traumatic amputation of foot, heel or lower third leg	12	Rt Side –8 Lt Side –4	24%
3	Partial traumatic amputation of foot, heel or leg	33	Both sides –6 Rt Side –14 Lt Side –13	66%

Total No of casualties/operation —50

Table 1 shows the types of mineblast casualties treated in the above mentioned period. 29 mineblast cases were received during 14 days war alongwith other battle casualties, and 21 cases were received subsequently upto 11 June 1973. Out of these cases, four cases were from civilian porters, and the rest were Army personnel. Average time taken for 29 cases to reach hospital from the time of injury was forty six hours, and for 21 cases the average time taken was twenty-two hours although the hospital is located within a reach of 5 to 8 KMs from the minefields all around. Out of 46 Army personnel injured due to mineblasts, there were 1 Officer, 1 JCO and 44 ORs.

Table 2 : Types of Surgery Done

Ser No	Types of Surgery	No	Reamputation at Field Hospital	Percentage
1	AK amputation	1	Reamputation after a BK amputation	**
2	BK amputation-unilateral	40	1 (AK amputation done)	80%
	BK amputation-bilateral	2	Nil	4%
3	SYMES amputation	2	Nil	4%
4	TRANSMETATARSAL amputation	2	Nil	4%
5	Amputation of toes	1	Nil	2%
6	Massive debridement	3	1 (BK amputation done)	6% **

Table 2 shows the different types of amputation done, for these cases. Massive debridement were done in three cases, two cases out of which had injuries to heel with severe degree of comminuted fractures of calcaneum and one out of these two needed reamputation. The third case surprisingly escaped injuries to his feet, but had multiple injuries over both legs. Cases needing amputation of toes or transmetatarsal amputations had relatively less damage to the tissues. One AK amputation (Reamputation), twenty-three BK amputations, two Syme's amputations, one transmetatarsal amputation, one case of amputation through toes and two cases of Massive debridement were done during 14 days war period. Rest of the amputations were done subsequently.

DISCUSSION

In twenty-one out of twenty-nine cases received during fourteen days war, limbs

---

** For % Reamputation not counted

were mangled, grossly contaminated and patients had severe oligaemic shock. Most of them were priority 1 cases and considerable time had to be spent for their resuscitation. Twelve of these cases reached hospital with impending gangrene, and one of them needed AK amputation 48 hours after a BK amputation, since the initial amputation failed to stop the spreading gangrene. In all these cases, the Hb% was between 5gm% to 10gm%. Maximum time taken for a mineblast case to reach the hospital was eighty hours and thirty minutes. The entire series of twenty-nine mineblast cases were resuscitated with Dextravan, plasma and infusion fluid, and only twenty-one bottle of blood could be procured and given. In six out of these cases, while changing the dressings 48 hours after 'local amputation', stumps were not too healthy. All these six cases had severe oligaemic chock, anaemia and also badly contaminated wounds. In all these cases amputations were at the lowest levels where the tissues were considered viable. Some, out of these six cases might have required further amputations due to wound conditions.

Out of twenty-one post war mineblast cases DPS was done in Sixteen cases with excellent results. This figure includes all the civilian patients treated. At the time of DPS, wounds were reasonably healthy. The time of DPS was between 72 hrs to 96 hrs. All these cases were resuscitated with plasma and other infusion fluids, only four bottles of blood were used in post war series. One case in this group treated initially with massive debridement of heel required a BK amputation just above the ankle. All the civilian amputees stayed in the hospital for four weeks in average, the delay in evacuation from hospital was solely due to the problems in rehabilitating them. However necessary arrangements could be made to procure prosthesis by sending one civilian patient to ALC, Pune, and the other to Ludhiana.

Early active movements of the joint above amputation for all these cases were stressed to prevent flexion-adduction contraction. Four cases with prosthesis could be followed up, no complications of amputation what so ever was seen in anyone of them.

Table 3 : Associated Injuries

Ser No	Associated Injuries	No	Percentage
1	Penetrating Injury chest	1	2%
2	Eye Injuries	1	2%
3	Traumatic Amputation Rt hand	1	2%
4	Soft tissue injuries of buttocks and thighs	4	8%

Table 3 shows the incidence of associated injuries in this series. One case was extremely complicated due to severe degree of haemopneumothrax on the left side due to penetrating injury chest. 830 ml of blood was aspirated from left pleural cavity. A water seal drainage was fixed which helped in re-expansion of left lung. In the same patient, a BK amputation was done under GA. Another case had blast injuries to both eyes in addition to injuries to lower limb, the third had simultaneous traumatic amputations of hand alongwith one foot. Other four cases had minor soft tissue injuries of thighs and buttocks in addition to blast injuries of feet/legs.

Ablation of limbs is unfortunately only too often required in surgery in the forward areas and mineblast injuries are the main contributing factors for this. The most distressing conditions presenting before the surgeon are those which result from delayed evacuation from forward defended locality, gross infection of already severely damaged tissues due to possibly factor beyond the control.

ANAESTHETIC PROBLEM

Traumatic surgery of limbs poses varieties of anaesthetic problems. The main factor which deteriorates the conditions of a patient is the delay in evacuation and institution of appropriate resuscitative measures. Further due to excessive influx of cases at a time, and due to limited resources available in forward-most areas, anaesthetist has got to assess the cases and decide the type of anaesthesia necessary under the circumstances. Problems are further aggravated due to excessive loss of blood resulting in low Hb% in blood. It is a common teaching that spinal anaesthesia is a contraindication in a case who has been in haemorrhagic shock. But such cases can be managed under spinal anaesthesia (under adverse circumstances with sudden influx of cases) after being stabilised by vigorous resuscitative measures. In the entire series of 50 cases, 15 cases were operated under mid-spinal anaesthesia in lateral position with a cotinuous I. V infusion. Rest of the cases were operated under GA. This helped in conservation of gases, drugs, saved the time, helped in manning two operating tables single-handed and dealing of cases which were unfit for GA. There were no immediate or post operative complications whatsoever in any case done under spinal anaesthesia.

For a surgeon, facing the problems in such cases during a war or even during peace time are no different than mentioned above. His decision has to be quick, bold and accurate in every amputation case, specially when he is already busy in tackling streams of various other types of casualties during a war. It is needless to say that an anaemic and badly dehydrated case is a potential danger for gross infection, delayed wound healing, even gangrene and subsequent reamputation.

It is always advisable to try only local amputation in such cases. Conservation of healthy tissue is bare minimum requirement but surely not at the cost of threatening gangrene. In all isolated cases, for an experienced surgeon there is nothing wrong in going for classical amputation where indicated but the skin flaps should be kept open or just poximated in the needle to be followed by delayed primary closure. Results of such cases in post war series were highly satisfactory, as there was no hurry for evacuation. A tourniquet no doubt helps in saving time, but not much of difference has been felt without tourniquet and not a single case in this entire series was operated under tourniquet.

CONCLUSION

In a snowbound area, where during a snow season the height of snow ranges between 30′ to 40′, the minefields pose special problems since the mines slide down if not anchored properly. It is obligatory that all minefields should be properly and accurately shown in the maps and troops in the particular sector be well conversant with it.

Evacuation of mineblast casualties should be given priority at any level, all basic principles of first-aid should be adhered to which include control of bleeding and early institution of resuscitative measures.

All emergency amputations should be at the lowest level at which the tissues are viable. When operation and evacuation conditions are unfavourable and for any reason and sepsis in wounds becomes a problem, then this rule must be adhered to. Above all, in all cases requiring amputation at any level, the surgeon must be satisfied that there is no hope on earth for the survival of the traumatised tissues which have been crushed beyond salvage.

ACKNOWLEDGEMENT

1. I am grateful to Lt Col S. K. Saha, AMC, Officer Commanding, 319 Field Hospital, for permitting me to publish the case reports.

2. My thanks to my colleague, Major R. K. Mehra, AMC, Classified Specialist (Anaesthesia) for help in preparing this paper.

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MAJOR GENERAL AMIR CHAND AWARD FOR 1974

The Maj Gen Amir Chand Trust Fund, which is administered and managed by the Director General Armed Forces Medical Services, awards a Cash or Cash & Medal Prize of Rs. 500/- annually for the best published work in a subject pertaining to any field of Medical Science, including clinical research. The term, 'Clinical Research' implies research into the mechanism and causation of disease, including its prevention and cure. It covers work on patient in hospitals and field studies in epidemiology and social medicine.

Medical officers of the Army Medical Corps, including those seconded to the Navy & Air Force, are only eligible to compete for it.

The Selection Board of the Trust Fund invites applications for the award of this Prize for the year 1974. Applicants must forward seven copies of their publications through proper channels to reach this Directorate General (DMR) by 01 Dec. 74. Only one article published by an officer during 1974 will be accepted. Work conducted under an approved Armed Forces Medical Research Committee (AFMRC) Project, in respect of which a final report has been published by the AFMRC during 1975, will also qualify for consideration of the award.

The prize for 1974 will be awarded at the XIII Meeting of the AFMRC scheduled to be held during 1975 at the Armed Forces Medical College, Pune when the recipient of the award will be given an opportunity to present his work.

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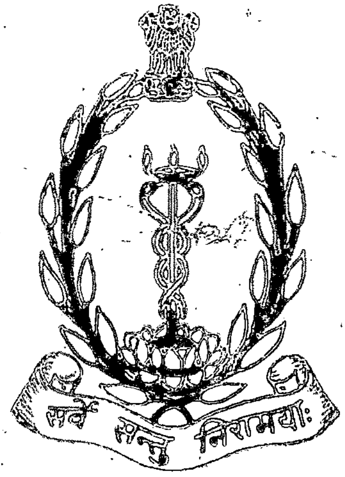

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BIFID NOSE

BY

Dr. M.M. Mukherji, MBMS (Cal), FRCS (Eng & Edin), FAMS*

Face develops from the following embryological processes between four and eight weeks of foetal life :-

(a) Frontonasal process, above.

(b) Two lateral processes, above and lateral.

(c) Two maxillary processes, below and lateral.

(d) The mandibular arch, below.

(e) Stomodeum and Stomodeal membrane centre.

Stomodeum is recognizable at 4 weeks' embryo. At 5 weeks the nasal pits develop as a depression between two folds in the neurocranial portion (Frontonasal process). Between 5th and 6th weeks the two folds develop into Nasolateral and nasomedial processes to include the nasal pit in between them. Thus frontonasal process differentiates into Medial nasal and lateral nasal processes, itself remaining in between the two medial nasal processes. At six weeks the nasooptic cleft has been formed between the maxillary process and the nasolateral process. The naso-lateral clefts are still not united.

At seven weeks almost all the clefts have closed, giving contour to mouth and external nares. The nasolateral process has joined with the maxillary process. The globular processes - downward projections of the nasomedial processes complete the upper lip by fusing with the maxillary processes.

Development of Nose : (Fig. 1–5)

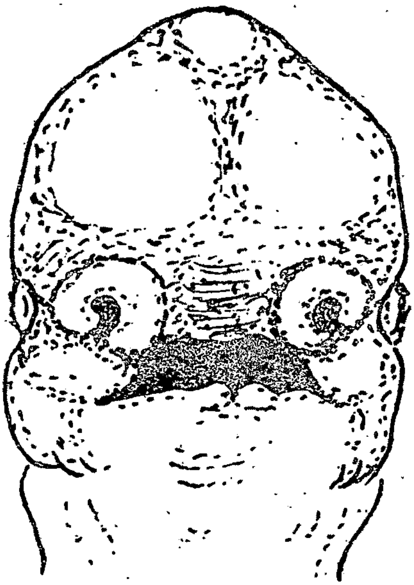
Nasomedial or median nasal processes which developed from the horseshoe-shaped ridges over the nasal pits grow downwards and towards each other. The two,

---

*Emeritus Professor in Plastic Surgery,
Institute of Post Graduate Medical Education and Research, Calcutta.

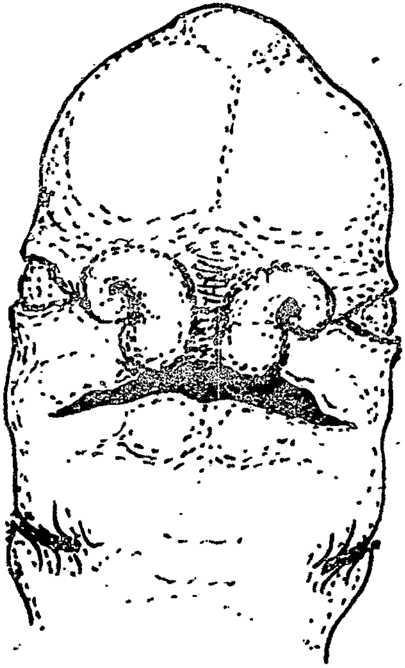
median nasal processes can only come closer and fuse with each other if the frontal part of the frontonasal process recedes upwards.

(Figures 1 to 5 are showing normal development of face and nose).



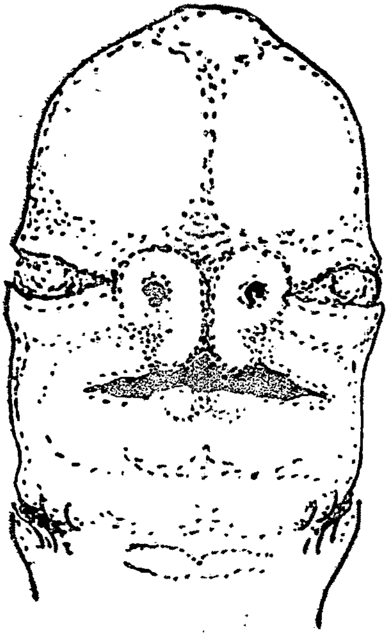
Five & half weeks.

Fig. 1



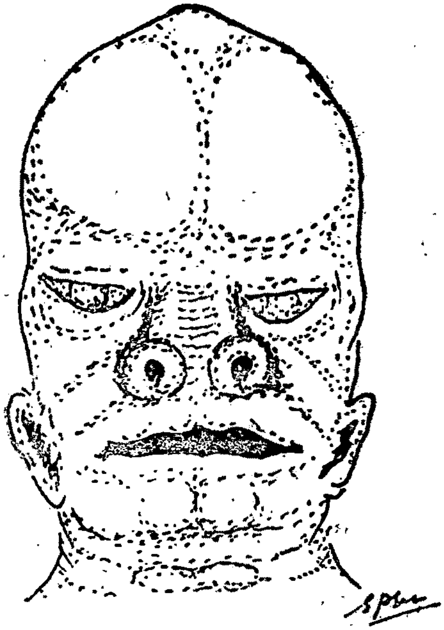
Sixth weeks.

Fig. 2



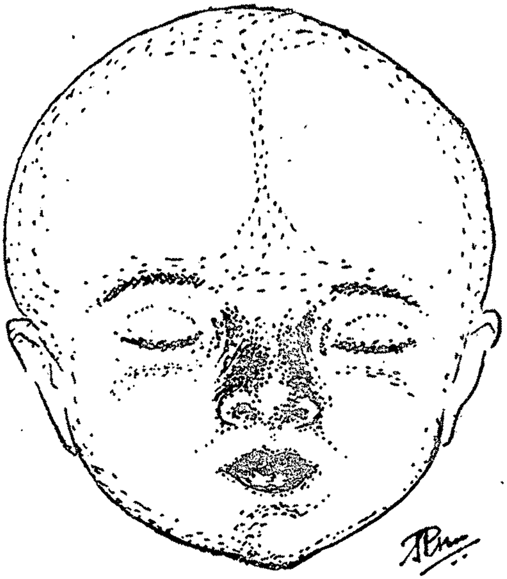
Seventh weeks.

Fig. 3



Eighth weeks.

Fig. 4



Normal

Fig. 5

Normally the medial nasal processes grow downward and towards each other encroaching on stomodeum. During this period, the broad frontal part of frontonasal process recedes upwards and gives chance to the medial nasal processes to fuse. The medial nasal processes then rotate inwards and disappear from the external aspects of the nose. The medial nasal processes form the septum of the nose. At its lower border the medial nasal processes send down the globular processes to form the middle part of the lip i.e. the premaxillary part (philtrum). When the medial nasal processes fuse they form the following :-

(a) Nasal septum
(b) Tip of the nose
(c) Columella
(d) Premaxilla
(e) Prolabium and philtrum of the upper lip

So it is realised that the nose develops from paired structures. These paired structures may be absent or may grow interrupted. They may even be reduplicated or may be completely absent. Failure of the two medial nasal processes to fuse may give rise to a rare median cleft of the upper lip.

Arrest in recession of the midline frontal process is the cause of separation of the nasal cartilages and bone in the upper portion of the nose. This also gives the deformity of hypertelorism. Failure in fusion of the medial nasal processes or the arrested growth of the medial nasal processes results in separation of two nostrils, deep grooving between the nostrils, reduplication of columella and lower septum and lastly the central cleft the upper lip. So due to defective fusion of the two medial nasal processes and due to non-recession of the frontal part of the frontonasal process, the following types of bifid noses are produced :-

(a) Broad root of the nose with some degree of hypertelorism. This hypertelorism may be apparently evident in severe degrees of flat noses. (Figs. 6-8).

(b) Wide, grooved and deformed nose with hypertelorism. (Figs. 9-11).

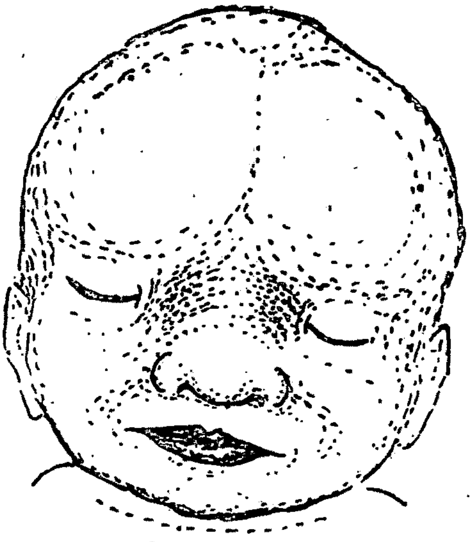
(c) Wide groove over the nose from tip upwards with a wide elevated dorsum of the nose. (Figs. 12 & 13).

(d) Minor forms of wide or grooved nasal tip with wide and/or grooved columella. (Figs. 14-16).

(e) Two completely separate noses. (Figs. 17 & 18).

These can be called as five degrees of the bifid nose and they are depicted in the following clinical photographs.

Fig. 6 shows the diagram of Group I.



(1)

Broad root of the nose

Fig. 6

Clinical features :

The deformities of the nasal bones, the nasal cartilages and the nasal septum are grotesque. When normal processes fail, the natural restriction of growth are lost so the nasal bones, septal cartilage and nasal cartilages take various shapes, develop into various sizes and create different angulations. The clinical entity may comprise of the following types of deformed noses :—

(a) Broad root and dorsum of the nose with wide gap in between the angles of the eyes (Hypertelorism).

This is caused by wide frontal spine or by extension of the frontal bone into the nose. In some patients with flat nose, there may be apparent Hypertelorism.

(b) Deformed, wide and thick nasal bone with wide septum.

The nose will feel like a hard irregular causing with bones and cartilages projecting in any and every angle. The septum is wide and consists of more than one piece of bone or cartilage. There may be extra pieces of bones, cartilages or fibrofatty tissues inside the septum (2nd and 3rd degree).

(c) Broad tip of the nose with or without a groove on the tip of the nose and/or on the columella. This is due to failure of fusion of the lower part of the two medial nasal processess (4th degree).

(d) Completely separate nose as shown in figure of the 5th degree.

Patients come for correction of deformities present from birth. As Plastic Surgery is getting more popular, smaller children are also being brought for corrective operations. Some of them have difficulty in breathing due to wide septum. But most of them come for a broad and/or a bifid nose.

Total number of patients - 13

Total number of patients treated in the Plastic Surgery Unit of the I.P.G.M.E. & R. and Howrah Railway Hospital in last 15 years is 13. As regards Sex distribution of the thirteen patients, seven were males and six were females.

Age distribution

0	—	10	...	4
10	—	20	...	7
21	—	30	...	2

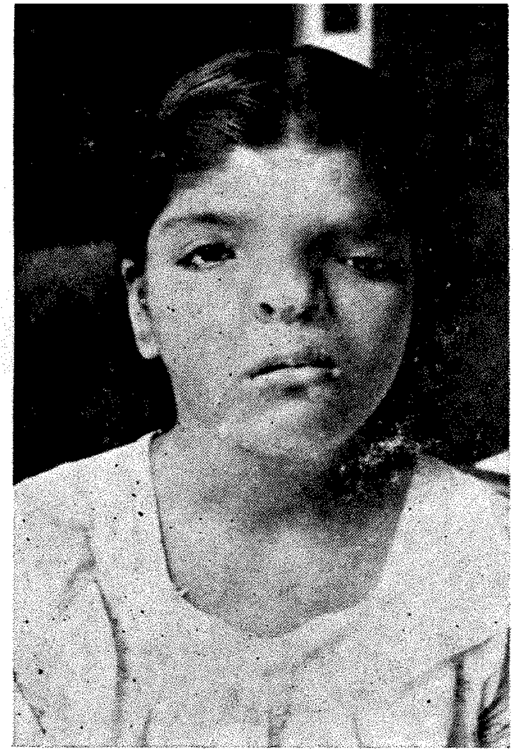


Fig. 7

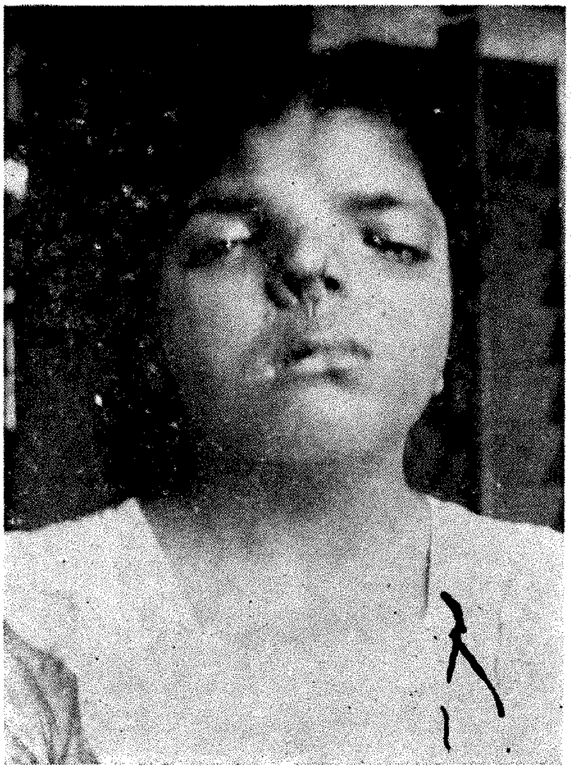
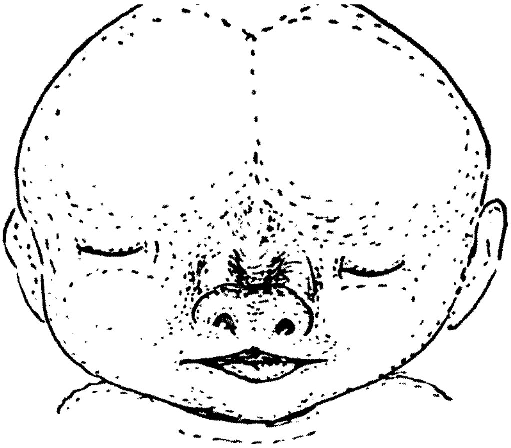


Fig. 8

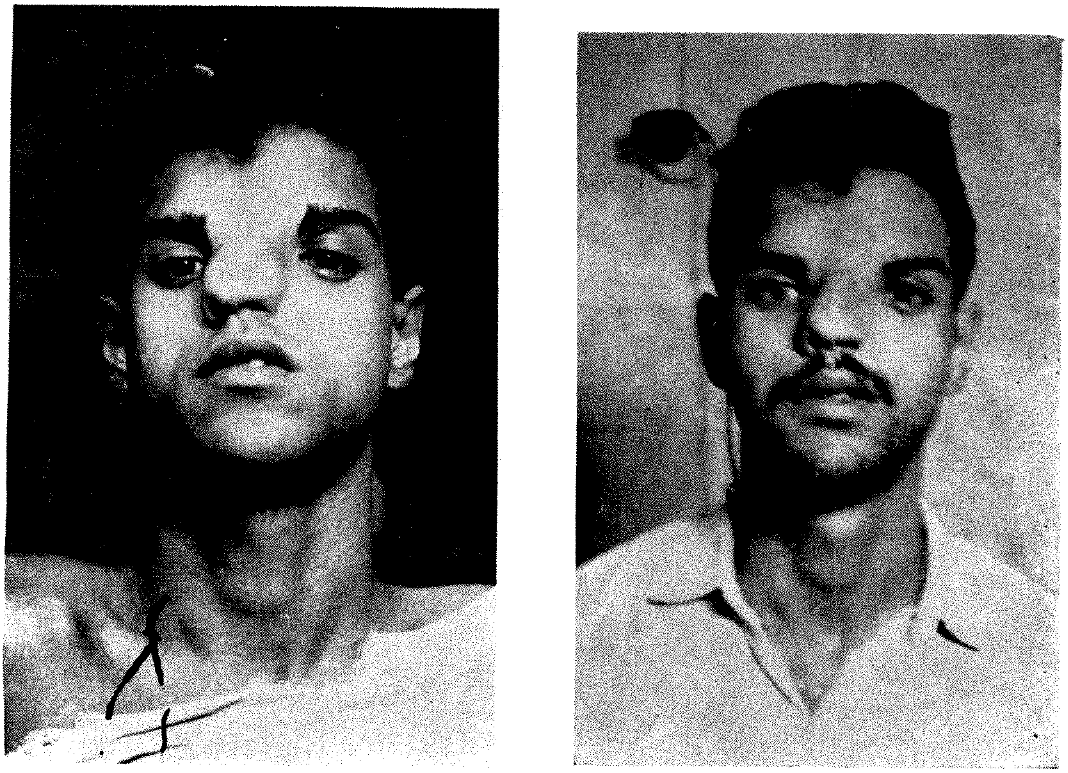


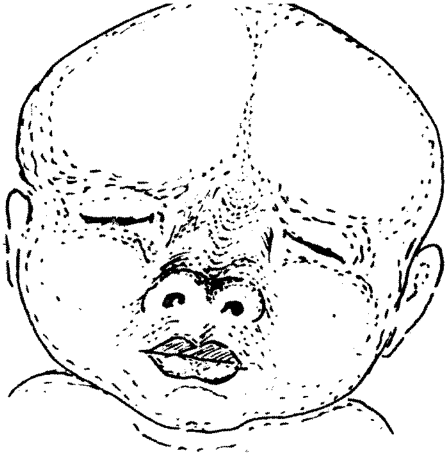
(2)

Wide, grooved & deformed nose
with hypertelorism.

Fig. 9

Photographs 10 to 11 show the actual incidences with result of treatment





(3)

Wide groove over the nose from tip upwards with a wide dorsum of the nose.

Fig. 12

Fig. 13 shows a photograph of Group III

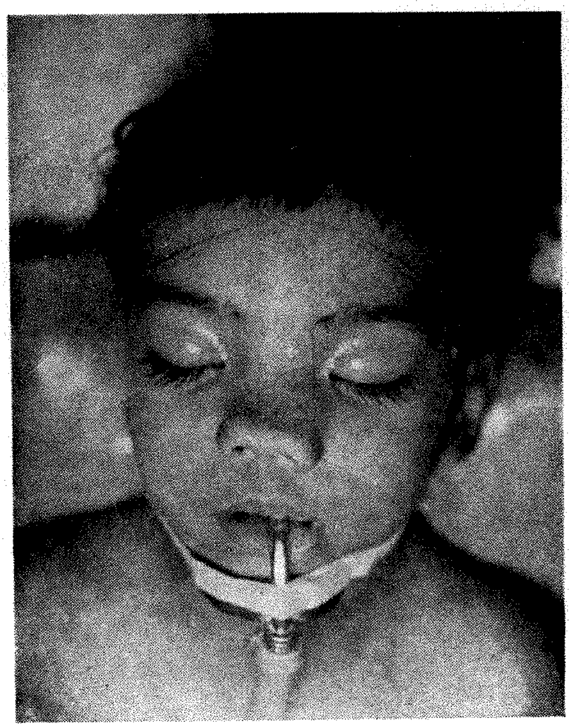
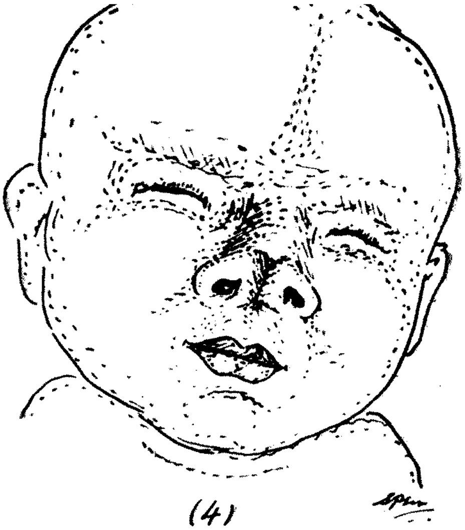


Fig. 13



(4)

Minor form of wide or grooved nasal tip with wide/or grooved columella.

Fig. 14

Photographs 15 and 16 illustrate Group IV, with result of treatment

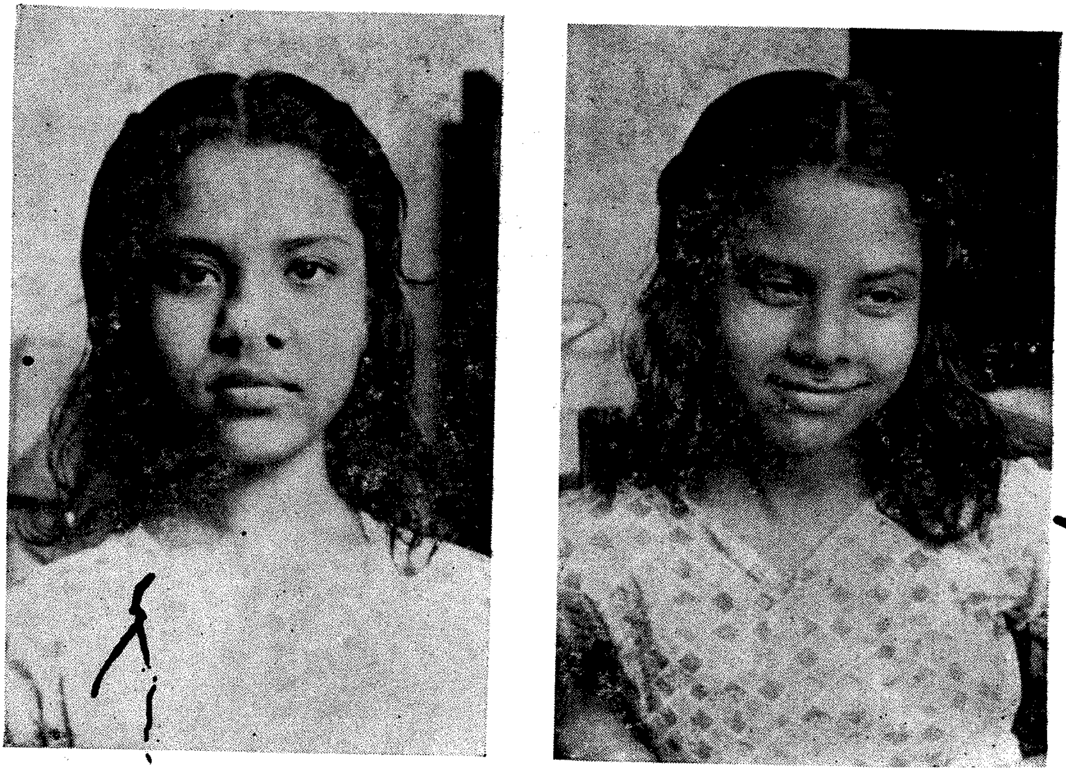
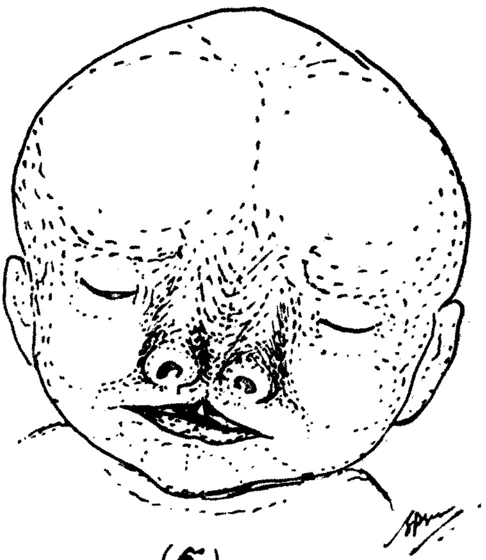


Figure 17 depicts Group V



(5)

Two completely separate noses.

Photograph 18 illustrates Group V

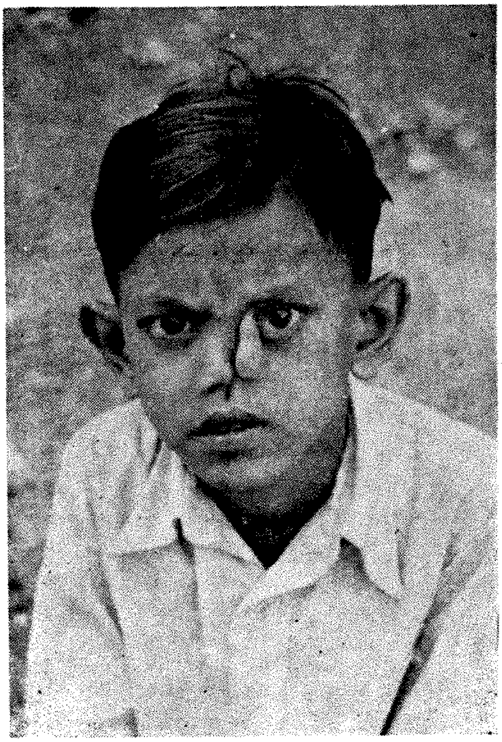


Fig 18

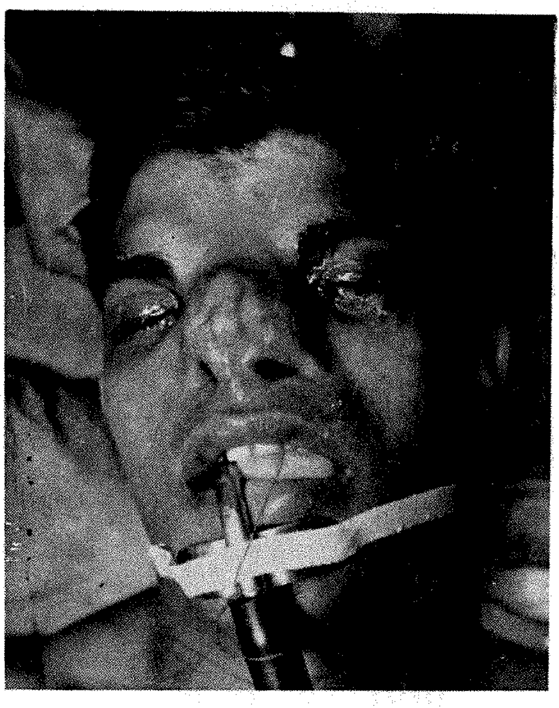


Fig 19

Photograph 19 illustrates the result of an intranasal unsuccessful operation

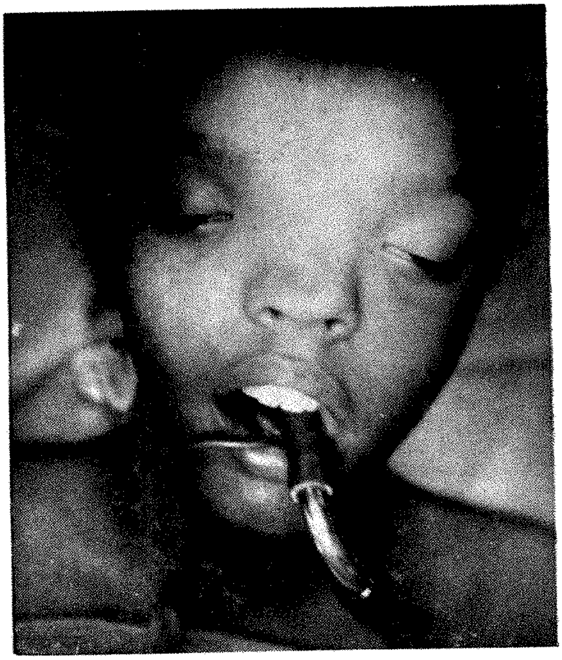


Fig 20

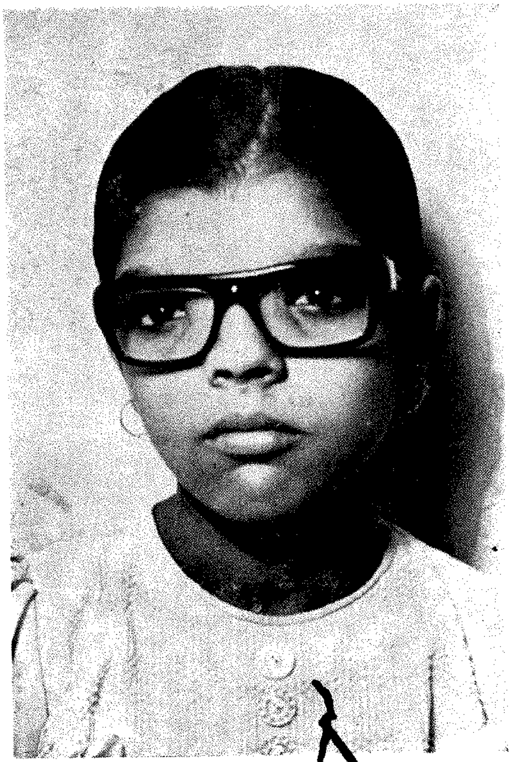


Fig 21

NEW OPERATIVE TECHNIQUE FOR HORSESHOE FISTULA-IN-ANO

BY

Dr. R.K. Menda, M.B., F.R.C.S., F.A.C.S., F.C.G.

An anal fistula is a tract lined by granulation tissue usually resulting from an ano-rectal abscess which burst spontaneously or was opened inadequately. The fistula continues to discharge either continuously or intermittently and seldom heals without surgical aid. This is because of repeated infection from the rectum or anal canal. 3.6

Various surgical procedures like chemical cauterisation of fistulous tract, laying open of fistula, fistulectomy in two stages, wide excision of fistulous tract, excision and skin grafting have been described by different authors. It was and is still believed that more the area excised the less the chances of recurrences. Skin graft usually fails because of infection and improper fixation of skin over the grafted area.

A new procedure for the treatment of horseshoe shaped fistulae is devised. The operative procedure is based on the fact that in majority of the case, the source of infection is the anal gland in the posterior midline and that most of the fistulae follow Goodsall's rule described below.

Goodsall's rule states that :-

(a) If the external opening is situated from an inch to an inch and a half distant from anal margin and either anterior to or on the transverse anal line, the internal opening will be found in the same radial line as the external opening and the main tract will be straight.

(b) When the external opening is situated at similar distance but posterior to the line, the interior opening will be found in the midline posteriorly and the tract will take a curved course.

(d) If the external opening is at greater distance than an inch and a half from the anal margin, whether anteriorly or posteriorly, then the internal opening will always be found posteriorly in the midline and the main tract will take a curved course backwards.

Anal glands open into the crypts of Morgagni at the pectinate line. They may be simple or racemose in type.[2] Simple ones are situated in the submucosa while the

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racemose type pass through the internal sphincter muscle and lie between the sphincteric muscles and conjoined longitudinal muscle. Number of glands varied from six to fifteen and are concentrated in the posterior circumference. Infection of these glands leads to abscess formation.[7]

At the onset, abscess may be local between the internal sphincter and longitudinal muscle of the rectum. The pus dissects through the longitudinal muscle into the superior posterior anal space passing either superiorly or inferiorly to the deep external sphincter.[4] Since both ischiorectal spaces communicate with the posterior superior anal space above the surface of the superficial external sphincter muscle, the pus will extend in one or both ischiorectal spaces. At this stage, if the abscess bursts, it will produce an anal fistula with the external opening posterior to the transverse anal line.[2] (Figs. 1 & 2.)

In some patients, the abscess extends to the perineum, labia and thigh and when it bursts or is inadequately drained, will form an anal fistula which extends anterior or transverse line, the trac. of which opens into the posterior midline internally. If such an abscess bursts at multiple places, it will have multiple external openings (Fig. 3). The primary opening however is single, situated in the posterior midline. Goodsall's rule is based on this pathogenesis. [5]

The New Approach :

The patient is put in a lithotomy position and fistulous tract is palpated. The internal opening is usually felt in the posterior midline in the vicinity of pectinate line. (Fig. 4). A midline incision of an inch and a half is made from the anal margin posteriorly. (Fig. 5). The external subcutaneous sphincter and the internal sphincter is divided upto the internal opening. The tract is then scraped from within outwards. (-Fig. 6). The external opening is excised and the tract is scraped. The tract and midline wound is packed lightly with ribbon gauze soaked in flavin and hydrogen peroxide. (Fig. 7).

When there are multiple external openings, each individual tract is scraped. The unhealthy margins of the external opening are excised.

If the external opening is anterior to transverse anal line, a counter incision is made in the posterior quadrant so that the entire tract can be scraped with ease.

One hundred and thirty-four cases of posterior semihorseshoe or horseshoe fistulae during the last three years are treated. Post operative hospital stay varied from twelve to fifteen days. The wound healed completely within fifteen to twenty one days. Diluted Hydrogen peroxide or normal saline was used for dressing the wound. Our follow-up has shown no recurrence so far.

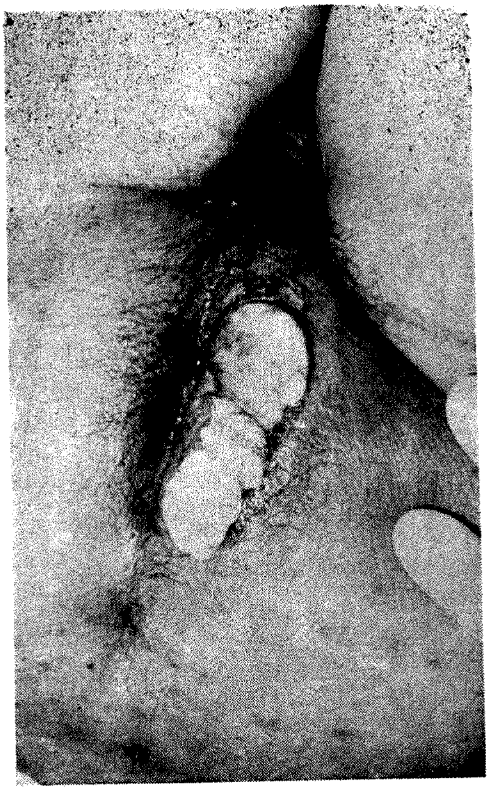


Fig. 1

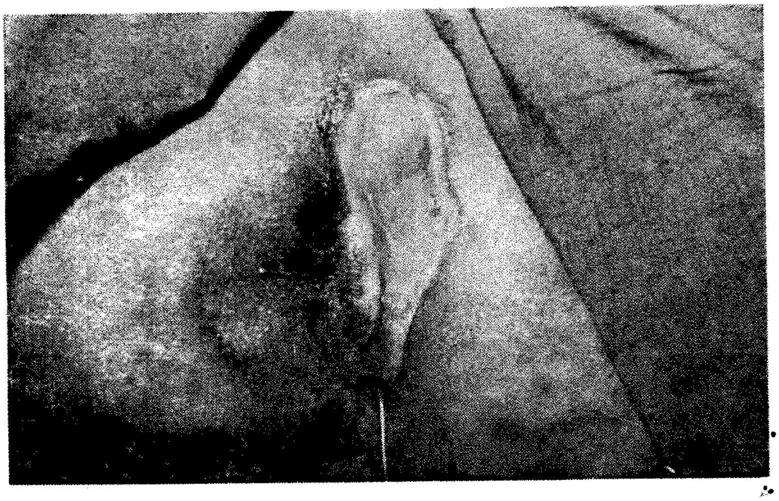


Fig. 2

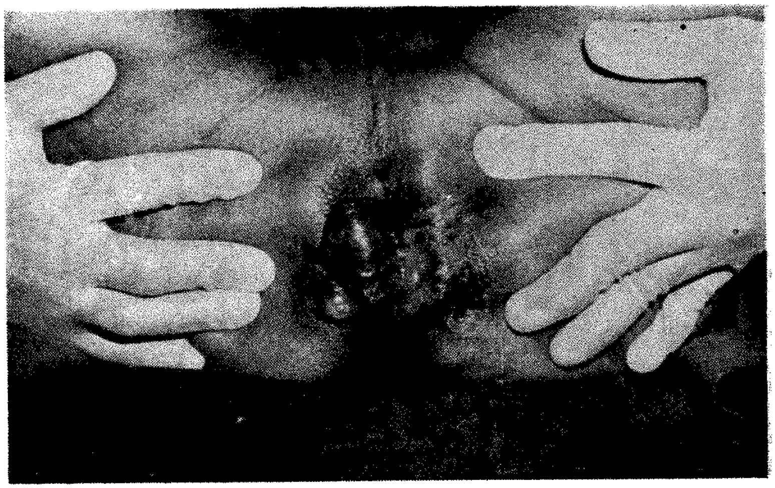


Fig. 3

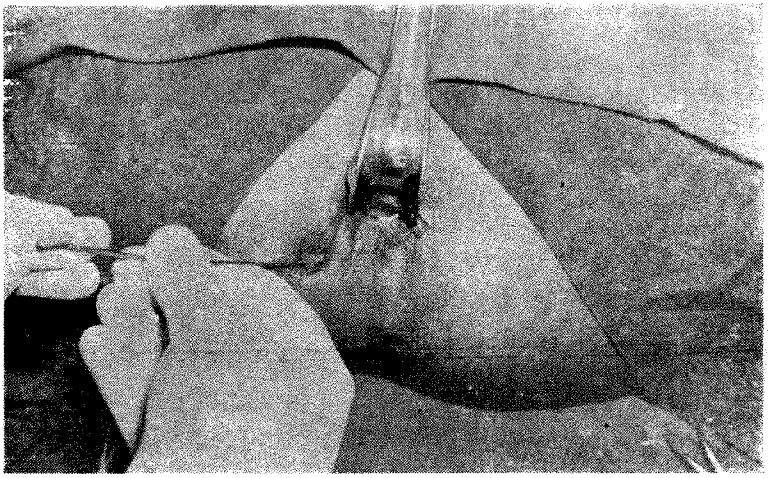
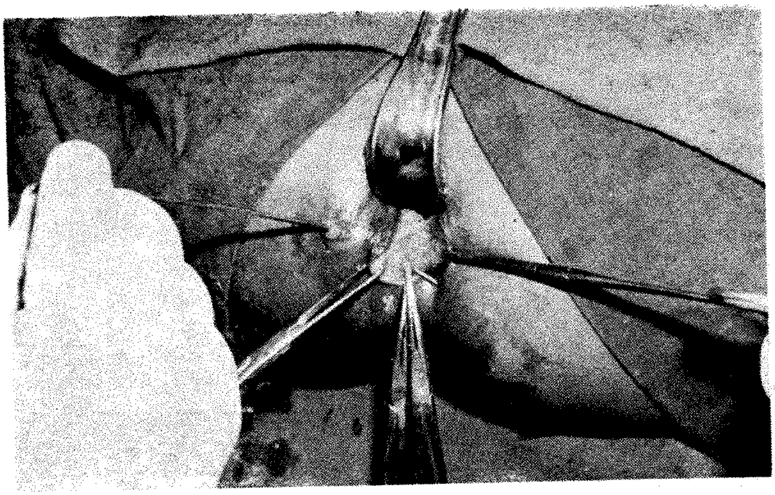


Fig. 4



Pig. 5

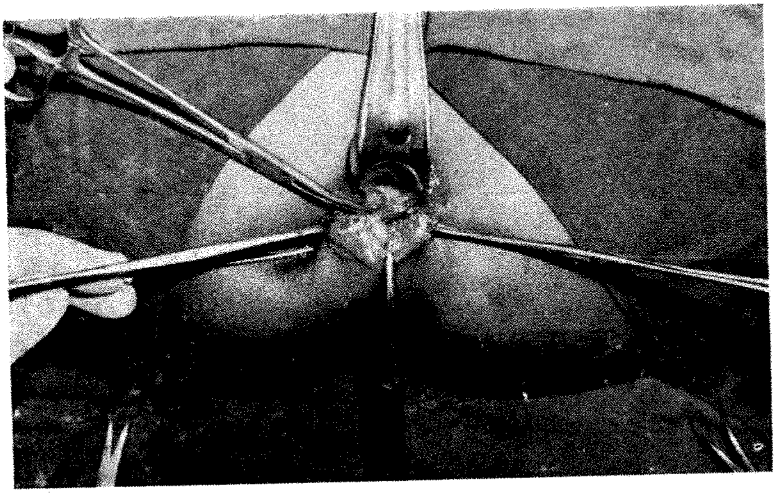


Fig. 6

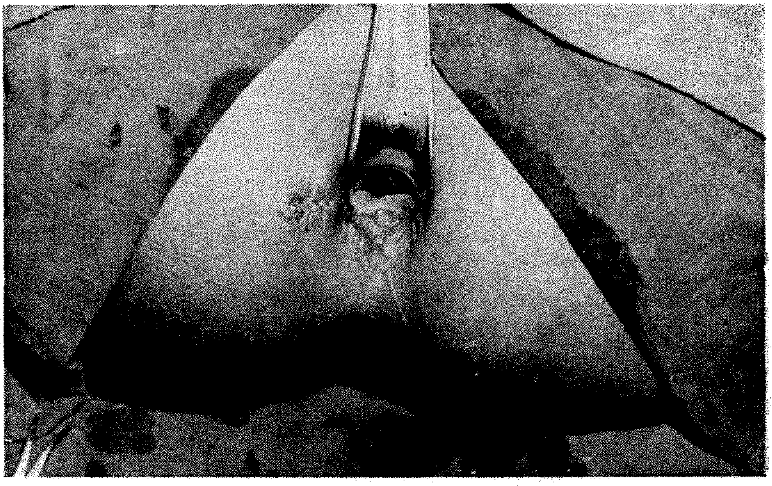


Fig. 7

The advantages of the new approach are :-

(a) It removes the source of infection, i.e. anal glands in the midline posteriorly at the site of the internal opening; thus removing the cause of the conditon.

(b) The resultant wound is small, linear and heals rapidly.

(c) Hospital stay is minimised.

(d) Undue scarring is avoided and there is no anal deformity.

(e) Incontinence does not occur after the operation.

(f) There is no recurrence.

All the fistulae were examined histologically. 115 were nonspecific in nature. Thirteen were of tuberculous etiology. Two were amebic in origin.

Table I

Semihorseshoe fistulae	89
Horseshoe fistulae	45
Nonspecific fistulae	115
Specific fistulae - Tuberculosis	13
Amebiasis	2

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WAR INJURIES OF THE HAND

BY

Lt Col P. M. L. Kathpalia*

INTRODUCTION

Hand is a universal instrument combining power with precision. Its functions depend upon delicate muscle balance, mobility of joints and exquisite tactile senstivity.

Functions of the hand are just as intricate as its structure but for simplicity they may be analysed into two basic types :-

(a) Pinch is a function of delicacy and precision. Essential requirements are an opposable thumb and at least one digit against which it can move.

(b) Grasp depends on mobility of fingers against a palm of sufficient breadth. This requires power. In a major hand injury it is seldom possible to preserve all the functions. Therefore, at the initial assessment, treatment must be planned, keeping these basic functions in mind. Aim should be to restore both or at least one.

Hand itself may be uninjured but may be crippled as a result of injuries to nerves, tendons or bones more proximally. It may also suffer by neglect.

MATERIAL

This paper is based on personal experience of treating 68 cases of injuries of the hand sustained during the recent Indo-Pak conflict. In addition 437 documents of hand injuries have been reviewed.

Analysis of cases

Table I

Mode of Injury

Shell	156
Grenade	9
GSM	171
Mine Blast	17
Accidents	83
Not known	1
Total	437

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Table II

Severity of Injuries

Minor (Abrasions small laceration etc.)	203
Major	234
Skin loss	9
Amputation	
Thumb	2
Single Digit	5
Multiple Digits	2
Single Phalanx	12
Tendon injury	
Extensor tendons	15
Flexor Tendons	10
Fractures	
Phalanges	91
Metacarpals	60

Table III

Duration of stay in Hospital

Less than 4 weeks	245
4 — 8 weeks	81
8 — 12 weeks	46
Over 12 weeks	65
Total	437

Medical Category on discharge from hospital

Category Aye	279
Category BEE	11
Category CEE	82
Final cat not known (Sick leave/Annual leave)	65
Total	437

7

MANAGEMENT

Aim of initial treatment of Hand Injuries is to procure primary wound healing without infection and minimal scar formation.

Principles which govern the treatment are :—

(a) Thorough surgical toilet in a bloodless field.

(b) Immediate primary skin cover.

(c) Prevention of infection by the above and chemotherapy.

(d) Early mobilisation.

Wound Closure:–Immediate skin cover is the most important single factor in the initial treatment. Repair of deeper structures is best left till after the wounds have healed and resolution of scar taken place.

Primary closure is the method of choice when the edges can be opposed without tension.

Free skin grafts may be used where primary skin suture is not possible and whe tendons, bones and joint capsules are not exposed.

Finger tip injuries may be treated in a variety of ways but without shortening of the digit as far as possible. We have used cross-finger flaps in most cases.

Extensive skin loss with injury to deeper structures requires full thickness skin cover which may be obtained by local re-arrangement of skin i.e. rotation or transposition flaps or by direct abdominal acromio-thoracic flaps. Cross arm flaps are considered ideal by some but they cause immobilisation of both the arms resulting in obvious inconvenience.

Treatment of Fractures :–In open wound reduction of fractures offers little difficulty, but maintenance of position in the presence of extensive soft tissue damage poses a problem. If conditions permit and facilities are available, Kirshner wire placed longitudinally is the simplest means of fixation.

Mutilating Hand Injuries :–Pose complex problems regarding assessment and planning of treatment. In general it may be said that the attempt should be to salvage a functioning hand with basic functions in view. It is possible to lay down certain guide lines.

(a) Preserve all possible length of the thumb. Where all other digits are lost a useful hand can still be salvaged.

(b) In multiple finger injuries, preserve the length of each remaining digit. Mobility of fingers is more important on the radial side and length on the ulnar.

(c) Preservation of the breadth of the palm adds to the strength of the grip.

Tendon Injuries :–Tendon involvement in war wounds is very common. Either they are injured by the missile, or involved in subsequent fibrosis. The former requires repair by direct suture or tendon graft. The later require tenolysis.

Surgery of tendons is undertaken only after good skin cover has been obtained, resolution of scars occured and joint mobility restored. Without going into details we have followed the principles formulated by Bunnel.

Restoration of Sensation :–Power and movement to be useful must be accompained by a good tactile sensation.

It has been our practice to repair digital nerves whenever found divided. Results are very rewarding.

Alternatively sensation may be restored by neuro-vascular island flaps.

Restoration of power in the paralysed hand :–Peripheral nerve injuries are common in wounds of the upper extremity. In cases where either nerve suture has not been possible/or has failed appropriate tendon transfers can restore the motor power of the hand to a large extent.

Immobilisation and Physiotherapy :–Although restoration of mobility as early as possible is the prime object of treatment, there are dangers in over-enthusiasm for early function. The importance of immobilisation for optimum healing though universally recognised in fractures, is often ignored in soft tissue injuries. All wounds of the hard should be immobilised during the initial healing phase. Plaster of paris slab accurately moulded to the contours with the hand in position of function is ideal. After wound healing has occured, movements must be started as early as possible.

CONCLUSION

The established principles of surgery of trauma are best illustrated in their application to the injured hand. Their application could not be more rewarding nor their disregard cripple so readily as in the case of injured hand.

7

PENETRATING WAR INJURIES OF THE CHEST

BY

Lt Col R.N. Thareja*

INTRODUCTION

During the recent Indo-Pak War a forward thoracic centre for the Eastern Theatre was established at Command Hospital Calcutta. The team consisted of a thoracic surgeon and a single ORA trained in thoracic surgical work. The experience gained during the management of this personal series forms the basis of this report.

MATERIAL

During the period 14 Dec 71 to 7 Jan 72, a total of 50 cases of penetrating injuries of the chest were treated at this forward thoracic centre. The material does not include superficial chest injuries or any cases of closed trauma of the chest. The patients were soldiers of the Indian Army, the Mukti Bahini, and some POWs from the Pakistan Army, who had sustained intrathoracic injuries in Bangla Desh. The mode of evacuation was by air to a Base Hospital from where they were driven for 15 miles before reaching this centre. The interval between the time of injury and arrival at this centre varied between 36 hours to 28 days, the average being 10.5 days.

The causative agent responsible for the intrathoracic damage was a bullet in two-thirds of the cases and shell fragment in one third. Multiple bayonet wounds were the cause in one case.

The majority of cases were of isolated chest injuries. However 4 cases had thoraco-abdominal injuries and two had associated brachial plexus injuries. Those with severe injuries of the head and spine have not been included in this series.

During evacuation from the front line to this centre some initial treatment had been received by all these cases, including treatment of the wounds. In 2 cases a limited thoracotomy had already been performed and in another 9 an intercostal tube had been inserted for closed drainage of the chest.

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PATHOPHYSIOLOGY

The basic pathophysiology in these 50 cases included pneumothorax in 2, haemothorax in 21, haemopneumothorax in 22, traumatic pneumonitis in 2 and empyema in 2 cases. 19 cases had one or more foreign bodies retained in the body (Table I).

Table I

Pathophysiology in 50 Penetrating war injuries of chest

Nature of injury	Number of cases
Pneumothorax	2
Haemothorax	21
Haemopneumothorax	22
Traumatic pneumonitis	2
Empyema	2
Retained F.B.	19

The traumatic pneumonitis referred to here was a result of the tissue damage caused by the high velocity missile during its passage through the lungs. (Fig 1).

OPERATIVE PROCEDURES

Major thoracotomy was performed in 10 cases for evacuation of clotted haemothorax, decortication of lung and removal of foreign bodies. In another case urgent thoracotomy was required for control of continued haemorrhage. Intercostal drainage in 4 cases was peformed for haemopneumothorax, and 2 ribresections were done for drainage for empyema. 12 cases of haemothorax required repeated aspiration. Simple pneumothorax in 2 cases was treated by a single aspiration in each instance. In 19 cases no surgical procedure had to be performed at this centre. Operations performed at this centre are given in Table II. Those performed elsewhere before arrival at this centre have not been included.

Table II

Operative procedures performed in 50 cases of Penetrating war injuries of chest

Operative Procedure	Number of cases
Major Thoracotomy	11
Intercostal drainage	4
Rib-resection	2
Thoracentesis - repeated	12
Thoracentesis - single	[illegible]

COLLEGE OF

MANAGEMENT

As the number of cases was manageable and all surgical facilities were available, it was decided to provide definitive surgical treatment at the forward thoracic centre itself. Important therapeutic measures included adequate blood replacement, avoidance of infection by administration of antibiotics, early surgery, where indicated, and active physiotherapy.

The aim of treatment was early restoration of normal respiratory function, achieved by obtaining clear bronchial passages, empty pleural cavities, well-expanded lungs and a stable mobile chest wall. Avoidance of oversedation so as not to depress the cough reflex, efficient physiotherapy, early ambulation, and bronchoscopy, whenever indicated were the measures undertaken to ensure patency of the bronchial passages.

Haemothorax in the early stages when blood is still in the fluid stage, can usually be treated by complete aspiration, repeated every 24 to 48 hours when required, till full expansion of the lung is obatined. (Ahuja 1966). In this series 12 cases of haemothorax were treated by repeated aspiration with good results. The lungs expanded fully in all cases, and none of the cases required any further surgical procedure. Intercostal tubel drainage may be resorted to if the haemothorax fills up repeatedly. However, appropriate choice for the site of insertion of the tube and the constant nursing care to keep it functioning cannot be over emphasised. A tube inserted at a wrong site or a blocked tube not only defeats its own purpose, but also carries the added risk of introducing infection.

Clotted or loculated haemothorax, however, cannot be emptied out by needle aspiration or tube drainage. In such a situation early thoracotomy was performed for evacuation of clots and decortication of the lung. (Figs. 2A, 2B, 2C).

Simple pneumothorax when small was managed by needle aspiration. Insertion of an intercostal tube was performed when continuous air leak was detected. Haemopneumothorax with an air leak was similarly treated by an intercostal tube. In the case illustrated in Fig. 3A, a haemopneumothorax was noticed in a very ill patient in whom an abdominothoracic shell wound had been dealt with earlier by a laparatomy, removal of spleen and repair of a colonic perforation and a tear in the diaphragm. An intercostal tube accomplished early expansion of the lung (Fig. 3B and 3C). The shell fragment seen in the posterior costophrenics sulcus was removed at a later date when the patient was ambulatory and in a much improved general condition. (Fig. 3D)

The metallic foreign body responsible for the intrathoracic injury may be retained in the body. In this series retained foreign bodies were removed from the neck, abdomen, chest wall, lung substance, pleural cavity, and the mediastinum (Fig. 4). All

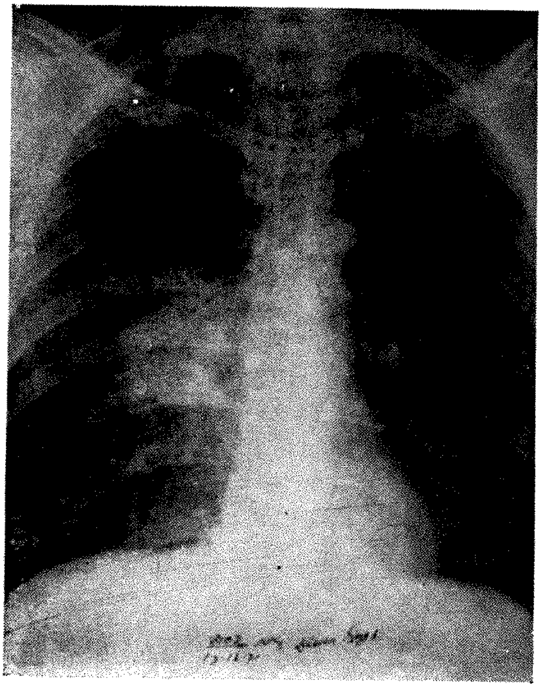


Fig. 1 Traumatic pneumonitis due to GSW chest. Note the opacity due to the lung damage caused by the high velocity missile.

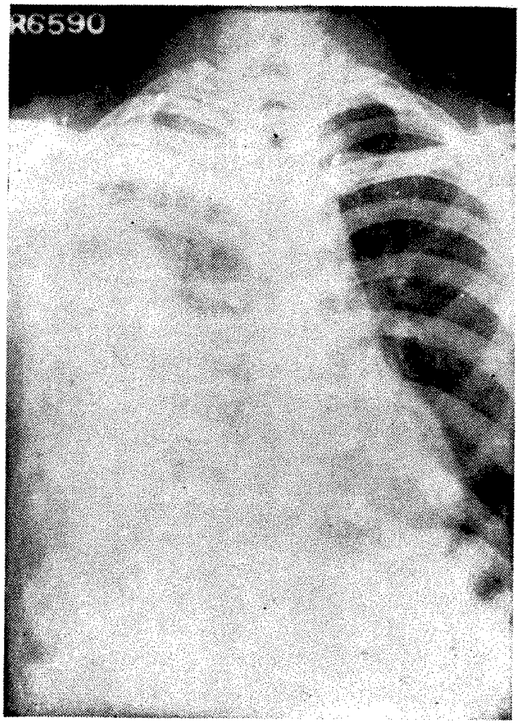


Fig. 2-A Clotted haemothorax due to GSW Chest. PA view.

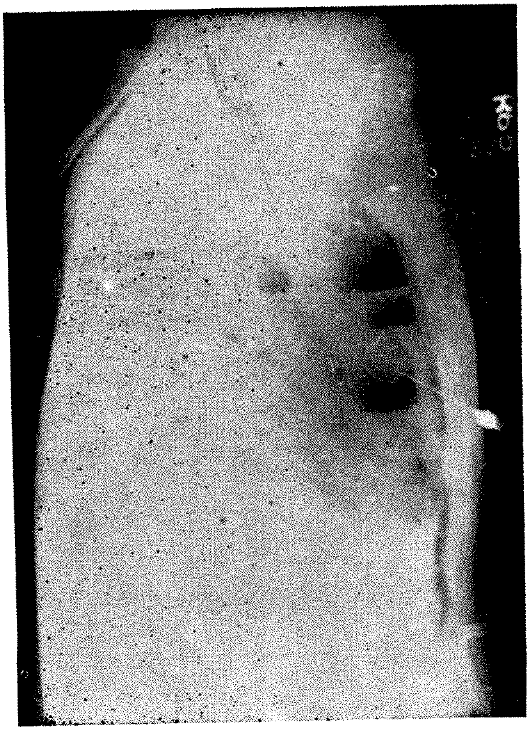
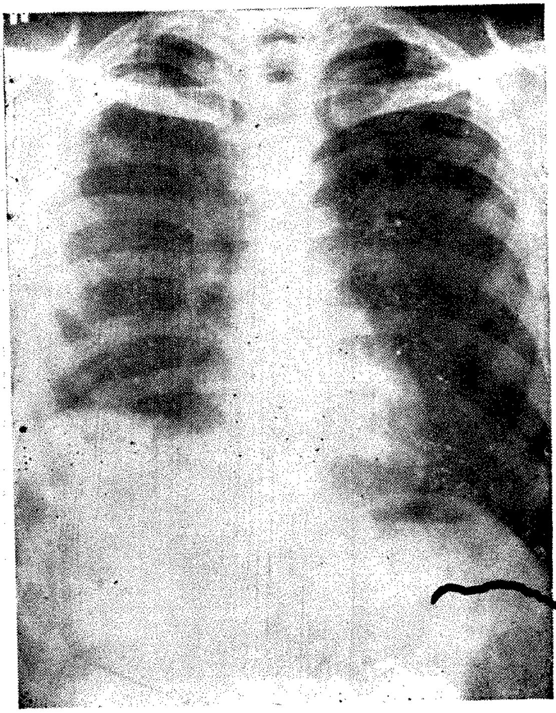


Fig. 2-B Same. Lateral view. The multiple fluid levels denote the loculations.

Fig. 2-C Same after thoracotomy for evacuation of clotted haemothorax, decortication of lung, and removal of F.B.



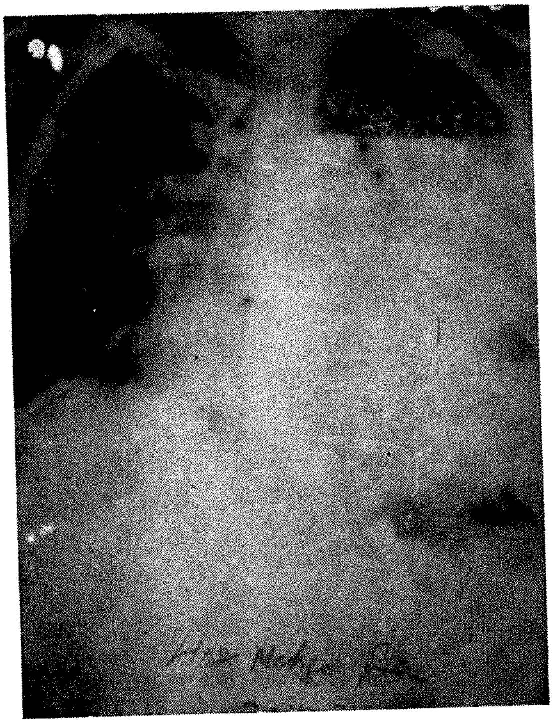


Fig. 3-A Haemopneumothorax discovered after laparatomy in an abdominothoracic injury due to a shell fragment.

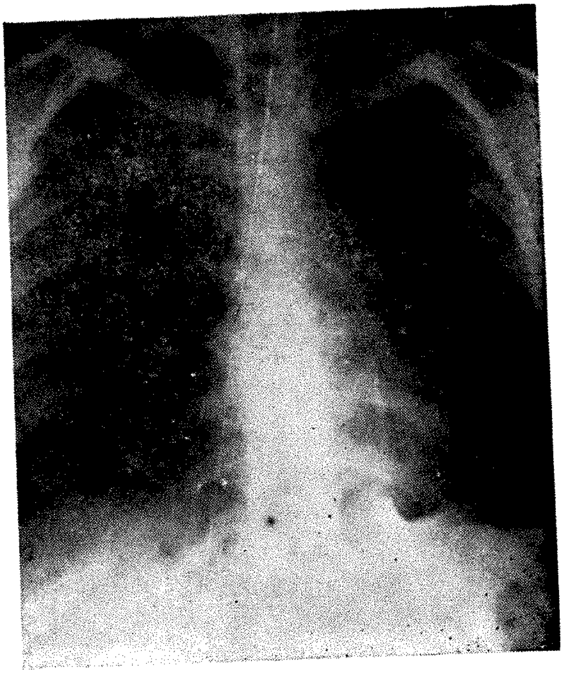


Fig. 3-B Same after intercostal drainage. PA view. Lung well expanded.

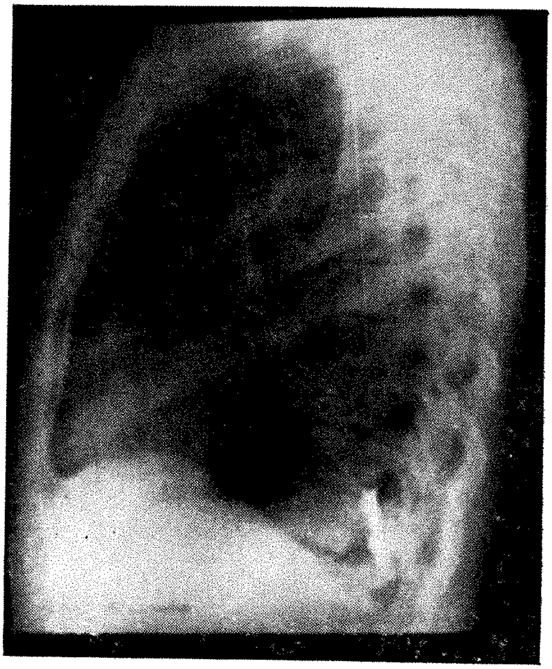


Fig. 3-C Lateral view. The shell fragment is seen in the posterior constophrenic sulcus.

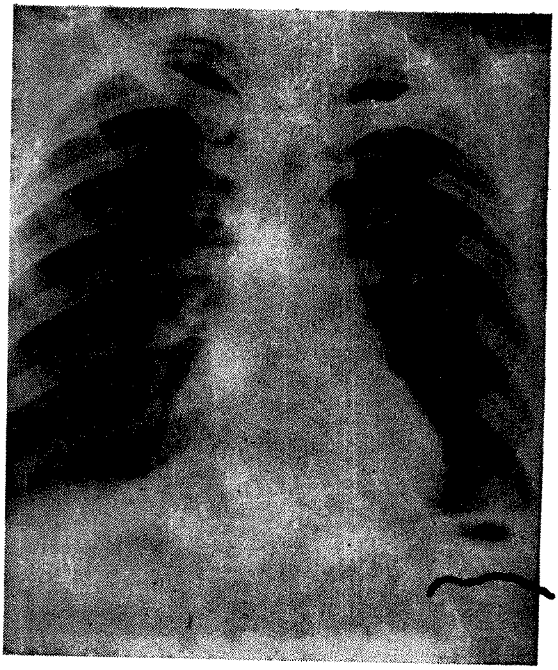


Fig. 3-D Same, after removal of the shell fragment.

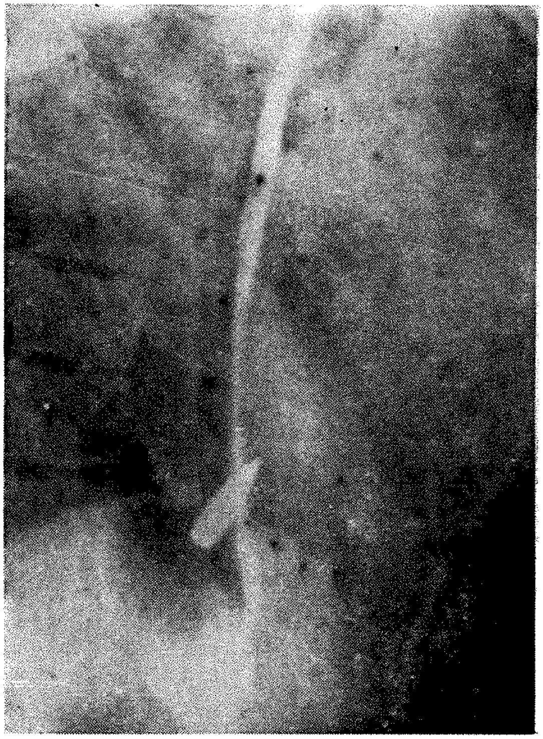


Fig. 4 Retained F. B. in the mediastinum (Lateral view). The bullet is seen overlying the barium filled oesophagus.

intrathoracic foreign bodies over 2 cm x 1 cm were removed irrespective of their location. Smaller foreign bodies were removed if located in the proximity of pulsatile vascular-structres or hollow viscera.

RESULTS

In the 50 cases of penetrating chest injuries treated at this centre there were no deaths. No patient required invalidement out of service due to the chest condition. The majority of patients left the hospital within two months.

COMMENT

The commonest problem was either a haemothorax or a haemopneumothorax. Haemothorax uncomplicated was managed by repeated aspiration. Clotted haemothorax required thoracotomy. Haemopneumothorax was easily managed by tube drainage.

The low incidence of empyema is noteworthy. The incidence of empyema in Korean war was 26%, and in Viet Nam war it had been brought down to 6% (Patterson 1968). In our small series it is 4%. Furthermore even these 2 cases were amongst the prisoners of war and had developed empyema before their medical care became our responsibility.

CONCLUSION

Positioning a thoracic team, well forward at a well equipped hospital, made it possible to provide definitive treatment at an early stage and thus effected a reduction in the period of hospitalization.

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A CRITICAL REVIEW OF THE MANAGEMENT OF CHEST INJURY CASES IN THE 1971 INDO—PAK WAR

BY

Lt Col AM Ahuja*

The 1965 Indo-Pak conflict produced 467 chest injury cases which were reviewed and commented by me. During the 1971 Indo-Pak War there were 488 cases of chest trauma which have again been reviewed by me.

In the present series, out of 488 cases, 251 had sustained an internal injury, the rest being superficial. There were 53 deaths, out of which 38 cases were either brought in dead or were in a moribund state on arrival and hence beyond surgery. Thirteen cases died soon after surgery due to severe initial injury and life saving surgery was not successful. Another 2 died due to late complications. All of the survivors, except 4 cases, have since been returned to military duties. This leaves no doubt that this time, when compared to the last, our performance has been much better.

The pathology in vast majority after chest trauma has been a haemothorax or a pneumothorax or a combination of the two. The measures available to relieve these conditions are three—

(a) Chest aspiration.

(b) A tube insertion with under water seal (closed thoracotomy).

(c) A formal thoracotomy.

Suggestions to improve our performance still futher are as follows :—

(a) Forward surgeons situated as they are in less equipped places should *not* conduct a formal thoracotomy. In contrast to a laparotomy, a thoracotomy needs greater skill and large amount of blood. In this series, it was felt that 3 deaths occured by attempting a thoracotomy under adverse conditions. The forward surgeon should rely on lesser procedures and evacuate the patient.

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(b) A tube should be inserted into the chest when the main pathology is a pneumothorax or as an adjuvant to closure of a sucking of the chest. While a tube can also be used beneficially at a static hospital in cases of haemothorax, insertion of a tube in cases of haemothorax in the forward areas is not advisable because the chances of its mis-management are very great and it was observed that at least in 8 cases an empyema resulted in this series due to faulty management of the chest tube.

Tube insertion became popular after the American surgeons published their results of the Viet Nam War. But it should be noted that these superior results were due to the fact that all their cases were very fresh when the blood in the chest was still liquid and also they used 2 tubes with electrical suction. The procedure made it possible for the lung to expand quickly and they were able to remove the tubes in 2-3 days before the tubes had the chance to infect. Such facilities do not exist in our forward areas, hence lungs do not expand so quickly and the tube carry the infection into the chest.

A tube is also not advised when a haemothorax is over 7 days old since in most such cases the blood already gets clotted. tube is not able to bring the clot and an empyema results. The correct treatment for clotted haemothorax is the evacuation of the patient o a thoracic surgical centre where a thoracotomy can be performed.

(c) Lastly, if and when there is a next war, by which time it is expected that we will have many more thoracic surgeons trained, the thoracic surgical teams should be positioned immediately in selected hospitals to afford expert treatment to patients with minimum delay. This will help improve our results still further.

ACKNOWLEDGEMENT

I am thankful to Lt Gen AK BARAT, MVC, PVSM, Chief Consultant in Surgery for sending the material of thoracic injuries to me for analysis.

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UROLOGICAL PROBLEMS IN INDO-PAK WAR 1971

BY

Major Mohan Kumar*, Capt (Miss) S. R, Rao**
Lt Gen A. K. Barat, PVSM, MVC, PHS***

Wounds of the urogenital tract during World War I, their incidence and problems in management have been reported by Young. Culp and Kimbrough presented their experience of injuries of Genito-urinary tract in European theatre of operations during World War II. Busch in 1967 reported 38 cases of urogenital wounds at Vietnam war, out of a total of 370 wounds and treated at United States Naval Hospital.

Twenty-two patients with urogenital injuries were evacuated to Army Hospital Delhi Cantt, during the period December 1971 to June 1972. All patients were from Western Sector except one who sustained ureteral injury in Bangla Desh operation. This forms 2.3 percent of a total of 958 wounded received at Army Hospital during Indo-Pak war in 1971. Most of these patients had received primary definitive surgery at various forward hospitals. In addition, four patients of acute renal failure were received. The experience gained from these 26 cases with various urological problems is presented in this study.

Incidence

The incidence of urogenital injuries in various operations of war is shown in Table I. In World War II, Marshall reported these injuries in less than 1 percent of patients wounded in field and Patton in 2—4 percent. Busch noted unrgenital injuries in 10 per cent of Vietnam casualties received at United States Naval Hospital. In the present study only 2.3 percent of all casualties treated at Army Hospital had injures of genito-urinary tract.

Table I

Incidence of Urogenital Injury

ORLD WAR II	
Marshal	1%
Patton	2 — 4%
VIETNAM SERIES	
Busch	10%
INDO-PAK WAR 1971	2.3%

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Site of injury

Statistics regarding the site of injury and the cause of injury in 22 urogenital wounds have been analysed and compared to those of World War II as reported by Culp and Kimbrough and World War I by young (Table 2 and Table 3).

Table II

Site of Injury

Site	Indo - Pak (Authors)	Vietnam (Busch)	World War II (Culp)	World War I (Young)
Kidney	22.5%	20.8%	30.0%	36.7%
Ureter	9.0%	4.2%	3.8%	2.2%
Bladder	4.5%	8.8%	16.3%	31.7%
Urethra	18.0%	6.3%	15.0%	
Penis	18.0%	25.0%	14.0%	
Testis & Scrotum	27.0%	35.4%	21.0%	

Table III

Cause of Injury

Injury	Patients	% OF TOTAL
Shell Wound	12	54.0
Gun Shot Wound	7%	31.5
Mine Blast	1	4.5
Grenade	1	4.5
Crush Injury	1	4.5

Renal Injuries

In our series, five patients with missile injury to the flank and abdomen were explored at forward hospitals for intraperitoneal visceral injuries. Rupture of kidney was detected in them during operation and nephrectomy was done in all (Table 4). In Vietnam series, only one of ten patients with renal injury had nephrectomy and this was necessary due to severance of renal vessels.

Table IV

Renal Injury

Opeaation of War	TOTAL	Nephrectomy	
		No.	%
Vietnam	10	1	10%
Indo-Pak 1971	5	5	100%

Ureteral Injuries

Two patients had ureteral injury which were missed during primary abdominal exploration at forward hospital and they developed urinary fistula (Table 5). In both the cases it was possible to salvage the affected kidneys. Ureteral re-implantation into bladder was done in one case and the other patient had total ileal replacement of ureter since the middle third of ureter was missing (Fig. 1).

Total V

Ureteral Injury

Operation of War	TOTAL	Kidney Salvaged
Vietnam	1	1 (100%)
Indo-Pak 1971	2	2 (100%)

Bladder Injury

There was only one case of extra-pritoneal rupture of bladder due to shell wound. Suprapubic cystostomy and repair of bladder was done at Field Hospital. This patient recovered fully.

Urethral Injuries

There were 4 cases of urethral injuries. Two had only urethral contusion involving bulb due to fall on perineum and suprapubic cystostomy was rightly done in field. In one of them an attempt was made after a few days to repair the urethral injury by rail-road method under wrong assumptian of complete rupture of membranous urethra. Urethrogram done at Army Hospital showed false stricture of bulbous urethra and urethral dilatation cured both these patients.

One patient had incomplete rupture of membranous urethra due to fracture pelvis. He was initially managed by Foley catheter drainage. Voiding cystourethrogram at Army Hospital revealed stricture involving membranous urethra and a large false diverticulum communicating with the bladder anteriorly suggesting a leak from the extra peritoneal part of the bladder. The diverticulum was laid open by retropubic abdominal dissection and was cauterised. The stricture was repaired by the technique of Badenoch pull through urethroplasty. The patient achieved normal stream of urine and is free from urethral dilatation. Post operative urethrogram showed normal urethral calibre (Fig. 2).

Another patient had complete rupture of membranous urethra associated with severe fracture dislocation of pelvis. A suprapubic cystostomy was done in field and a few months later the stricture was repaired at Army Hospital by Badenoch urethroplasty with excellent result.

Penile Injuries

There were four cases of injury to the penis. In one patient, there was rupture of corpora cavernosa with a large haematoma which was evacuated and repaired with good functional result. / In the other case, who had bilateral orchiectomy in field, the penile skin was completely lost and this was repaired by split skin graft. Other two patients had minor penile injuries.

Testis and Scrotum

In this study six patients had injuries to testis and scrotum. Five of them had bilateral orchidectomy in field. This is considered unfortunate. In Vietnam series, there were 10 cases of testicular injury but no patient had bilateral orchidectomy (Table 6).

Table VI

Testicular Injury

Operation Of War	TOTAL	Bil. Orchidectomy
Vietnam	10	Nil
Indo-Pak 1971	6	5 (83.3%)

Acute Renal Failure

Four cases of acute renal shut down were evacuated to Army Hospital. A patient who developed anuria due to mismatched blood tranfusion, survived with medical management and did not require dialysis. The other three patients were received in moribund state with hyperkalaemia and blood urea 300 mgm. All of them died. These patients were not given peritoneal dialysis at forward hospitals and could not be given haemodialysis at Army Hospital.

DISCUSSION

The intense fighting for a short period of 14 days in the Western Sector during Indo-Pak war 1971 resulted in large number and varieties of Indian casualties. The care and ultimate survival of an injured patient depends on the rapidity with which he reaches a well equiped specialised surgical centre. Although the evacuation during this war has been more efficient and rapid as compared to previous conflicts, and significant number of patients were evacuated to Army Hospital by air, there has been a time lag varying from 6 days to 6 months before the patients with urogenital wounds were received at Urology Centre. In Vietnam war similar casualties received primary definitive surgery at the field hospital and with-in a few days were flown by jet transport planes to the United States for specialised treatment.

Every efort should be made to conserve the kidney and nephrectomy should be done when the renal pedicle is severely damaged or the renal parenchyma is irrepairably torn. When abdominal exploration at forward hospital reveals a ruptured kidney, the clots should be removed and attempt made to repair the parenchymatous tear by interrupted catgut sutures. This alone will control the bleeding effectively in most of the cases and the time thus gained can be utilised for rapid evacuation of the patient to a specialised centre where proper assessment of the renal injury can be made with investigations like renal scan, high dose excretory urogram and renal angiogram. This will help considerably in adopting a conservative approach with an aim to preserve renal function.

It is generally agreed that ureteral injuries are missed during abdominal exploration but the diagnosis becomes evident when the patient develops frank urinary leak from the wound. In them the kidneys can be salvaged by ureteral reconstruction which should preferably be done at specialised centres.

It is felt that primary repair of ruptured urethra should not be attempted in field wher only suprapubic cystostomy and drainage of perineal haematoma or extravasated urine should suffice.

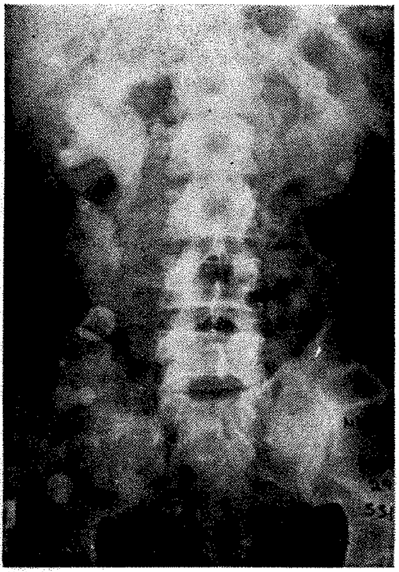

Fig. 1 A : Excretory urogram, in a JCO two months after gun shot wound to right flank who developed urinary fistula following abdominal exploration in field for associated visceral injury, shows moderate hydronephrosis on right side with extravasation of contrast from upper ureter.

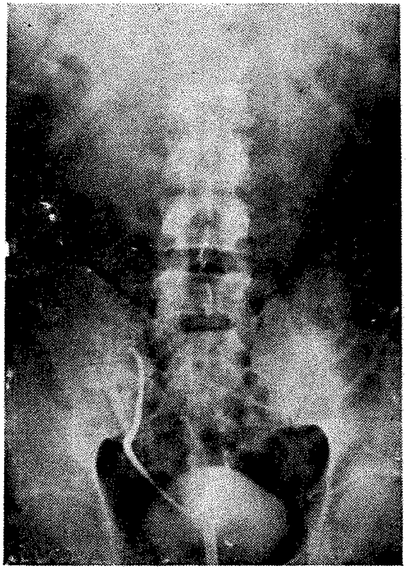

Fig. 1 B : Retrograde uretogram confirms that middle third of right ureter is missing

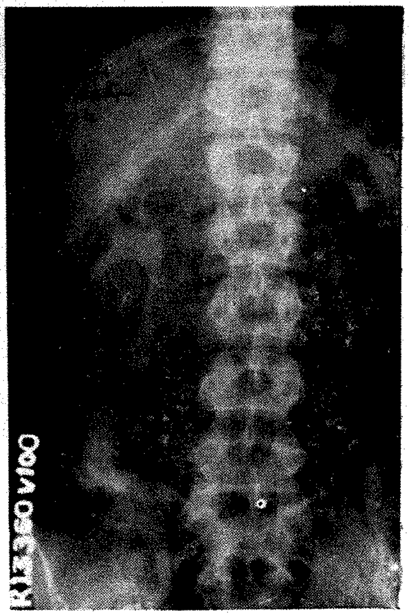

Fig. 1 C : Infusion pyelogram three months after ileal replacement of entire right ureter shows good renal function and complete regression of hydronephrosis

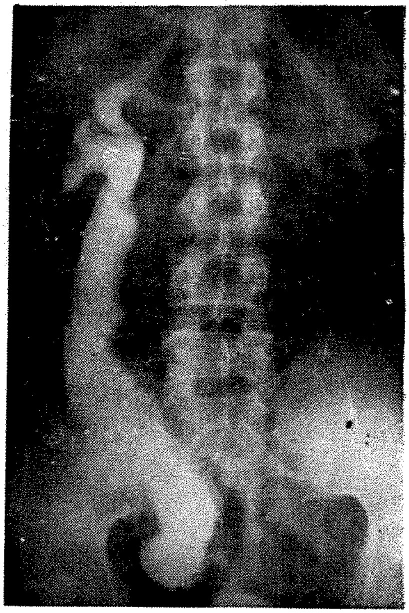

Fig. 1 D : Voiding cysto - urethrogram reveals vesico - ileal reflex which is a normal phenomenon after this operation

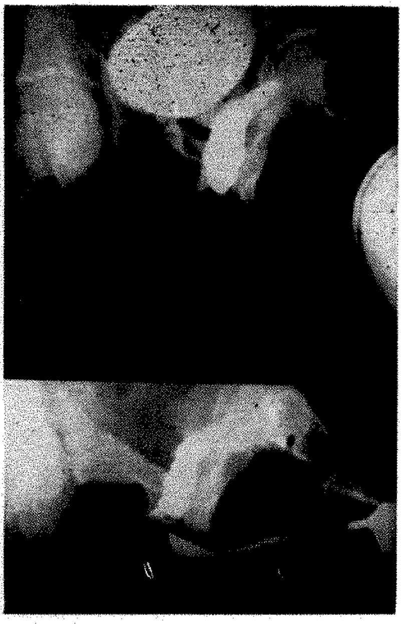

Fig. 2 A : Voiding cysto-urethrogram and retrograde urethrogram reveals stricture of membranous urethra with a large false diverticulum communicating with bladder anteriorly

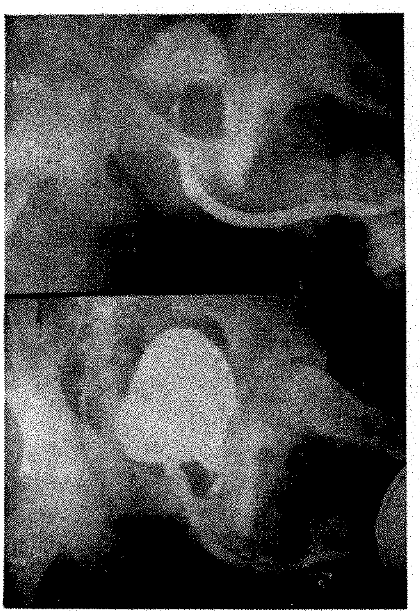

Fig. 2 B : Cystourethrogram three months after Badenoch urethroplasty shows normal urethral calibre

Bilateral orchidectomy should always be avoided. Attempts should be made to repair a torn testicle and it is worthwhile leaving behind a part of viable testicular tissue, which may avoid a life long harmonal therapy in a young patient.

Patients of acute renal failure with anuria should be given early and adequate peritoneal dialysis by the physician in any large Base Hospital. Some of them with hyperkatabolism due to severe trauma and infection with blood urea rise of over 30 mg per day will require rapid and early evacuation to a specialised centre for haemodialysis.

SUMMARY

Twenty-two patients with urogenital injuries were evacuated to Army Hospital Delhi Cantt during Indo-Pak war 1971. This figure accounted for 2.3 percent of the total wounded.

All five patients with renal injury had nephrectomy. It is felt that every effort should be made to conserve the kidney in forward hospitals.

Two patients had ureteral injuries and the kidneys have been salvaged in both.

In case of urethral injury, suprapubic cystostomy and drainage of perineal haematoma is all that is essential in field. Definitive urethroplasty procedure should only be done at a specialised centre.

Five out of six cases of testicular injuries had bilateral orhidectomy. This should rarely be required.

Patients of acute renal failure should be given early and adequate dialysis in an attempt to save young useful life.

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BLAST INJURIES OF THE EAR

BY

Brig Daryao Singh*

During the two Indo-Pak wars of 1965 and 1971, a total number of 133 cases of blast injuries of the ear were thoroughly examined and treated at Army Hospital Delhi Gantt and consequently follow up for atleast a period of six months.

Both the blast and noise trauma give rise to deafness and all these cases had varying degree of hearing loss affecting either the conducting or the perceiving part of the hearing mechanism. In addition, 77 cases had associated perforations of the tympanic membrane.

Tympanic membrane perforations can be closed by myringoplasty and middle ear machanism can be reconstituted to restore the conductive hearing loss but when the perceiving part i.e. the sensorineural region in the inner ear is damaged, it cannot either be relieved or cured and the hearing loss remains permanent. On the other hand, perceptive hearing loss caused by noise or blast becomes a major disabilitity with advancing age as it heralds an early presbycousis and adds to the disability of ageing deafness.

It thus becomes important that every effort should be made to prevent blast or acoustic deafnss. In advanced countries where such deafness is adequately compensated monetarialy, a serious thought is already being given to the preventive aspect of all type of acoustic and blast and aircraft noise or in the civil, by factory and machine noise.

At present, ear plugs and ear muffs are the only devices available for protection against noise or blast trauma. They work on the principle of excluding noise and blast by closing the external ear by firm pressure. This type of ear defender obviously has got its limitations of causing discomfort by too much pressure into the external ear. Efficient ear plugs and ear muffs do provide about 30 decibel attenuation of sound but have a further disadvantage of attenuating all type of sound and thus hamper communication.

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During the study of blast injuries of the ear resulting from the two Indo Pak wars, we at the Army Hospital Delhi Cantt, came across certain interesting findings which throw considerable light on the preventive aspect of noise and blast. These findings can be summarised as follows :—

(a) Pattern of hearing loss caused by blast effects is different from the normal accepted pattern of acoustic trauma. Blast deafness gives rise to a uniform hearing loss in all frequencies instead of dip at 4000 or 6000 cycles per second, so characteristic of acustic trauma.

(b) Sikh troops wearing turbans over their ears and armoured corps personnel wearing headphones for communication were protected against blast injuries by their ear covers. On the other hand, such devices offered little protection against acoustic trauma which is quite common amongst the armoured corps personnel.

(c) Blast effects are strictly directional and only the side facing the blast is affected, the opposite side escapes unhurt. Such absolute protection is not possible in case of acoustic trauma

In view of these findings, it was concluded that :

(a) a sharp distinction should be made between noise and blast effects on the ear.

(b) it is possible to protect the ears against blast without interfering with communication as in the case of a turban worn over the ears.

(c) in view of the strictly directional effects of blast waves, a multilayered porous material worn as a baffle over the ears would cut out blast but will allow sound to pass through and an ear defender of such pattern could be provided as a protection against blast.

These findings were reported in detail by us in the Journal of Laryngology and Otology (London). [2] Forrest and Coles[1] have also reported on a porous material in the form of an ear plug with small holes and they claim that such a plug has amplitude sensitive properties and it could cut out high intensity impulse noise while allowing lower intensity sounds to pass through. It is thus claimed to protect the ear against impulse noises without interfering with communication.

We thus had two sets of findings :-

(a) Our own clinical impressions resulting from the study of battle casualties that a porous material could protect against blast without interfering with communication.

(b) Forrest and Coles findings of a porous ear plug which could provide protection against impulse noise without interferin with communication.

We wanted to test these findings in the laboratory with a view to evolve an ear defender which will protect against both noise and blast without interfering with communication.

We therefore planned to carry out the following projects :-

(a) Laboratory experiments on blast to test various type of porous material which will not attenuate sound.

(b) Laboratory experiments on impulse noise to test porous material similar to that described by Forrest and Coles.

(c) Animal experiments on blast. In view of our clinical findings of war injuries we suspect that the site and nature of blast trauma in the cochlea is different from that of acoustic trauma.

(d) Field trials of the material developed by the above lab experiments.

Lab Experiments on blast were carried out at the Defence Service Laboratories in collaboration with Mr. Makhija.[3] Blast pressur created in a closed chamber was conducted through a blast tube on to the porous test materials and with the help of transducers, the shock strength of the blast was measured on Oscilloscope and photographed by poloroid film. Results of some of the materials are given in table 1.

Table I

Material	Blast attenuation
2 mm foam + Nylon net	44%
3 mm foam + muslin	63%
2 mm foam + 50 wire mesh	71%
3 mm foam + 25 wire mesh	87%
3 mm foam + 50 wire mesh	92%

These experiments have confirmed our clinical impressions from the examination of battle casualties and have further confirmed that it is possible to provide adequate protection of the ears against blast without interfering with communication.

Work on other projects is in progress.

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OCULAR WAR INJURIES (INDO-PAK WAR—1971)

BY

Col G. S. Sihota*

INTRODUCTION

Experience of ocular injuries during 1965 Indo-Pak War formed the basis for organisation and planning during the 1971 War. Some of the facts learnt during the 1965 War were as follows :—

(a) There was no incidence of intraocular infection.

(b) There was no incidence of Sympathetic ophthalmia.

(c) Quite a large number of enucleations/eviscerations were done in the field Area Eye Centres.

(d) A large proportion of intraocular foreign bodies (10 FB) were magnetic in nature.

(e) Vitreous haemorrhage in the absence of perforation absorbed within 2-3 months.

(f) Few cases of intractable traumatic iridocyclitis were encountered.

Based on the above facts, following conclusions were drawn :—

(a) Hasty intraocular surgery undertaken under far-from-perfect conditions in the Field Area is not justified in view of the complete absence of sympathetic ophthalmia and intraocular infection-the two most dreaded complications for which early surgery is generally recommended.

(b) Great emphasis should be laid on quick and direct evacuation of ocular casualties to Army/Command Hospitals. Rail travel is satisfactory for all types of eye injuries and one should not waste time waiting for air evacuation unless it was likely to materialise within a day at the most.

(c) **Provisioning of Special drugs/other materials :** Provisioning of the following items which could be very useful but are either not authorised or were not availale was done for Army/Command Hospital through the office of the DG AFMS :—

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(i) **Urokinase (Fibrinolysin) :** The only available drug in the world for dissolving blood clot in total traumatic hyphaema.

(ii) Fine corneal suturing material.

The above items were made available well before the war started.

ORGANISATION AND PLANNING

This war was neither so unexpected nor so sudden as that in 1965. This left enough time for organisation and planning which was done as follows :—

(a) **Proforma for ocular battle casualties :** A proforma was circulated to all ophthalmologists to be filled in respect of all ocular war injuries.

(b) **Evacuation, treatment and disposal of ocular casualties :** To cut short the transit period and to ensure best treatment, all Ophthalmologists were given guidelines as to which hospital the patients should preferably be sent depending upon the type of injuries.

ANALYSIS AND DETAILS OF ACTUAL WORK ENCOUNTERED

A total of 403 ocular injury cases were encountered. One third of the cases had other serious injuries besides ocular injuries thus admitted and treated in other wards while the remaining cases had only or mainly ocular injuries and were admitted to Eye ward.

All cases received at Army Hospital were given definitive treatment before being disposed off except reconstructive surgery where it could be delayed without detriment. Because of the pressure on beds, about 30 cases requiring hospitalization were transferred to CH (SC) Poona, MHs Roorkee and Bareilly. All others were either sent on sick leave or retained here till finality was reached.

OCULAR WAR INJURIES

(Indo-Pak War 1971)

ANALYSIS

Total (Minor-155; Others-248)	403
Bilateral Blindness	15
Unilateral Blindness	83 (29)
Perforated Injuries	110

Immediate Enucleations	56
Delayed Enucleations	14
Intraocular Haemorrhage	61
Traumatic Cataract	25
Conjunctivitis/Keratitis	148

CAUSES OF OCULER INJURIES

1.	Shells, Grenades, Bombs, GSWs	267
2.	Mines	49
3.	Others	87

TIME LAG

(To Reach Army Hospital)

(i)	Within 24 Hours	4 cases
(ii)	Within 72 Hours	20 cases
(iii)	72 Hours to 7 Days	All other

INVOLVEMENT OF EYES

Right Eye	Left Eye	Both Eyes
175	132	96

ASSOCIATED INJURIES

Ears	15
Others	83
	98

Important Clinical Features

1 **Intraocular Foreign bodies (IOFBs)**

(a) **Localisation**

(i) It was done with the aid of Limbal Ring X-rays. This simple method gave very satisfactory results.

(ii) It was further aided by Roper-Hall foreign body locator.

(b) **Treatment**

(i) Preoperatively magnetisability or otherwise of the foreign body was determined by the aid of Roper Hall Locator.

However this instrument although sensitive enough was not entirely infallible. Two IOFBs proved non-magnetic with the locator were sucessfully removed with the magnet.

We, therefore, operated on all cases of metallic IOFBs.

In our hands no harm has ever come because of the operation, even when foreign body was non-magnetic and thus not removed. This is based on long term follow-up of 1965 war cases.

(ii) **Posterior route extraction**

All IO FBs in this series were in the posterior segment. Posterior route extraction was attempted in all cases except one, incising the sclera as near the foreign body as possible.

2 GLOBE BURST (Effect of Blast)

This type of injury was much more frequent during this war than in 1965. In atleast 34 cases, the burst globe was the result of blast when a shell had burst very near and in front of the individual. There was no history of any fragments of the shell hitting the eyes nor any evidence of intraocular or orbital foreign bodies.

The bursts were situated a little outside the limbus but were not confined to any one sector. The burst area was approx. 15 mm x 5 mm with uveal prolapse and vitreous loss.

None of these eyes could be saved.

3 ORBITAL FOREIGN BODIES

There were several cases of such foreign bedies. No attempt was made to remove this even when foreign bodies were really large. All such eyes settled down without any evidence of orbital cellulitis.

4 CORNEAL PERFORATION

All such perforations were stitched with direct sutures using 5/7mm needles and 7-0/virgin silk. Air was injected into the anterior chamber in all cases afte washing out soft lens matter wherever indicated.

5 **Non Perforating Injuries with Intraocular Haemorrhage**

Almost all cases were given systemic corticosteroids for 4-6 weeks along with ten injections of entodon (Iodine preparation). This was to delay fibrous tissue proliferation and thus help in quick and complete absorbtion of vitreous and retinal haemorrhages.

6 Multiple Corneal Foreign Bodies

Only a few superficial ones were removed. Patients were treated with local antibiotic-cortisone ointment. Most of the remaining foreign bodies later sloughed out and the deeper ones proved to be completely inert.

Almost all such foreign bodies were sand stone particles which got imbedded in the face, lids, conjunctiva and cornea. These resulted from shell bursts near the individual.

7 Battle Neurosis/Cerebral Concussion/Flash Blindness

Three cases were received from battle field with history of complete blindness both eyes. Eye examination revealed no abnormality. There was, however, history of exposure to flash and blast as a result of shell burst near them. They regained normal vision within one week.

8 Mine Burst Injuries

These injuries were very extensive and very damaging. Besides causing bilateral blindness due to perforating injuries, one or both upper limbs were amputated and sometimes even the lower limbs.

9 Complications

(a) Infection, sympathetic ophthalmitis and Iridocyclitis.

There was no case of any of these dreaded complications. Mild to moderate degree of iritis and ciliary injection was, however, present in most of the injured eyes for variable periods.

(b) **Retinal detachment**

Three cases were encountered. In one detachment must have occurred at the time of injury or soon after, as at the time of operation subretinal fluid gushed out as soon as sclera and choroid were incised in an attempt to remove an intraocular foreign body.

CONCLUSIONS

(a) **Definitive Surgery**

This should be done in a base hospital having proper facilities and where the patients can be detained for sometime after surgery. Surgery in field hospitals including forward eye centrs should be confined to barest minimum like closing of large gaping corneal/scleral wounds.

Hasty intraocular surgery under far-from-perfect conditions in a Field Area is not justifiable in view of the fact that no case of intraoculor infection or sympathetic ophthalmia was seen during the 1965 war or during this war even when patients with perforating wounds with prolapsed uveal tissue reached the base hospital after a week of the injury.

Liberal use of antibiotic ointment locally and administration of systemic sulphonamides during transit proved effective in the prevention of sepsis.

(b) **Intraocular Foreign Bodies**

50% of the intraocular foreign bodies were found magenetizable. Roper-Hall foreign body locator although in perfect working condition was not found to be as sensitive as generally believed. Two foreign bodies which gave non-magnetic response on this instrument preoperatively were ultimately found to be magnetizable at operation.

(c) **Use of systemic corticosteroids**

Administration of systemic corticosteroids for 4–6 weeks starting immediately on receipt of patient and/or soon after operation reduced the incidence of severe type of iridocyclitis to nil. This seemed to have had no delaying effect on the healing of the wound.

We had a number of cases of intractable uveitis during the 1965 war when we had administered corticosteroids only when there was evidence of iridocyclitis.

(d) **Protective gear**

Almost 80% of injuries were caused by fragments of shells and/or by blast. Protection can be provided by the use of Protective goggles attached in the form of a sliding Visor to the helmet which should preferably be of fibre glass so that it is light in weight.

Worst injuries were those from mine explosions. In quite a few cases in addition to bilateral blindness, there was traumatic amputation of one or both upper limbs and sometimes even a lower limb. Personnel handling mines can be provided adequate protection by the following gear :-

(i) Chest guard consisting of strips of aluminium covered with cloth in the form of a waist-coat.

(iii) Gauntlet for the hand and forearm made of tough stuffed leather.

MANAGEMENT OF ABDOMINAL INJURIES IN THE FORWARD AREAS (PART II)

BY

Major U. Chattopadhayay, VSM*

I had the opportunity to work in a forward surgical centre which was giving cover to the heavy battle zones of Chhamb and Chicken neck during real trying condition between 3rd and 17th Dec 71.

Out of 303 battle casualties and battle accidents received in our hospital 16 were abdominal injuries. Out of 16 cases one had Thoraco-abdominal injury and another had perinio-abdominal injury. Thus abdominal injuries comprised of 5.3% of the total casualties.

Maximum number of casualties received in one day was 44. Out of 44 cases 2 were abdominal, one was chest, 18 other major and 23 minor cases.

Maximum number of abdominal cases in one day was 6 along with 5 others major and 7 minor cases.

Types of missiles and wounding agents :

Gun Shot Wound	– 10
Shell Wound	– 5
Crush Injury Abdomen	– 1

Time lag between injury and operation

Earliest was 2 hours; latest was 19 hours.

Condition of patients on arrival

Patient received in good general condition. (BP more than 100 mm Hg)	– 5
Patient received in moderate shock. (BP between 80 and 100 mm Hg)	– 3

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Patient received in severe shock (BP less than 80 mm Hg.)	– 2
Patient received in moribund condition (BP Pulse not recordable)	– 6

Pre-operative Resuscitation

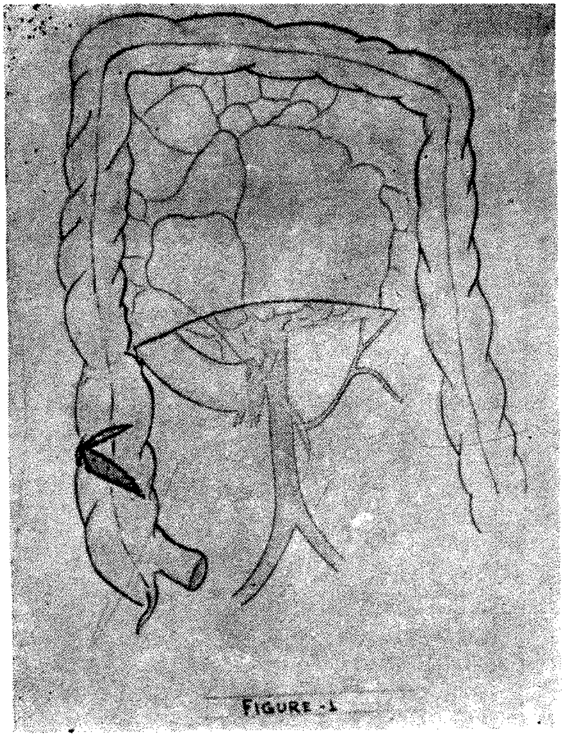
All were given blood transfusion ranging from 2 units to 7 units. On an average 2 litres of fluid in the form of blood, Ringers lactate, Glucose Saline and 5% Glucose were given. Other supportive measures like administration of morphine, antibiotics and Ryle' tube aspiration were carried out as a routine procedure. Where severe kidney or bladder injuries were suspected, catheter was passed for examination of the urine. If urine was clear, injury to the urinary tract could be excluded. Attempts were made to raise the BP to 100 mm Hg before operation. In 4 patients this optimum BP could not be achieved with any amount of transfusion but all were taken up for surgery though they were in moribund condition.

Types of injury, operative findings and technique

All of our series had multiple visceral injuries except in one case where only right kidney had partial coronal tear.

Analysis of Visceral Injuries

GI System	Genito-urinary System	Great Vessels	Associated Injuries
Stomach 3	Kidney Right - 2 Left - 2	Aorta – 1	Chest wall with lung- 1
Duodenum 2 Small Intestine 9		Splenic - 1	Major compound fracture- 3 Peripheral nerve injury- 1
Large Intestine : Right side – 2 Left side – 3 Mesentry – 2 Liver – 4 Spleen – 1 Pancreas – 2	Bladder – 2 Urethra – 1		Testis- 1



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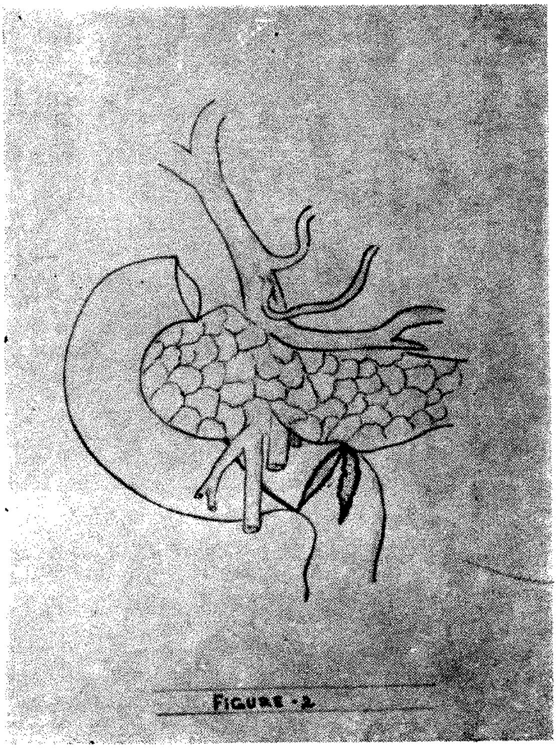

Fig. 2

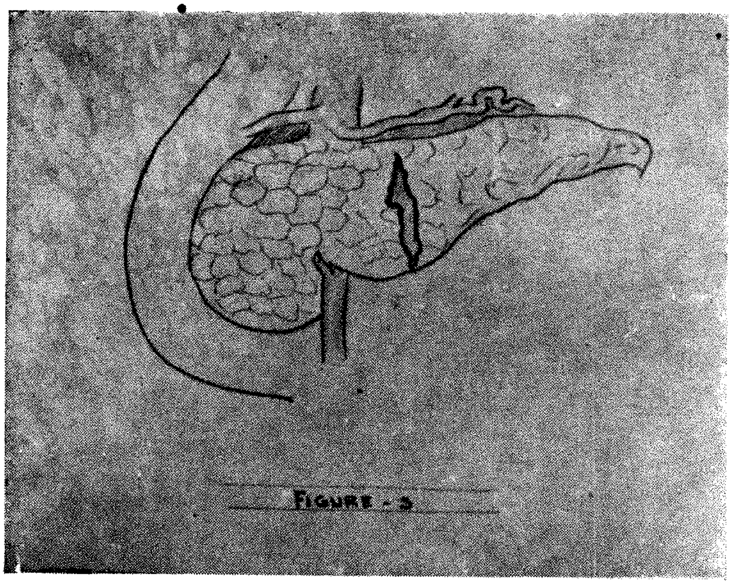

Fig. 3

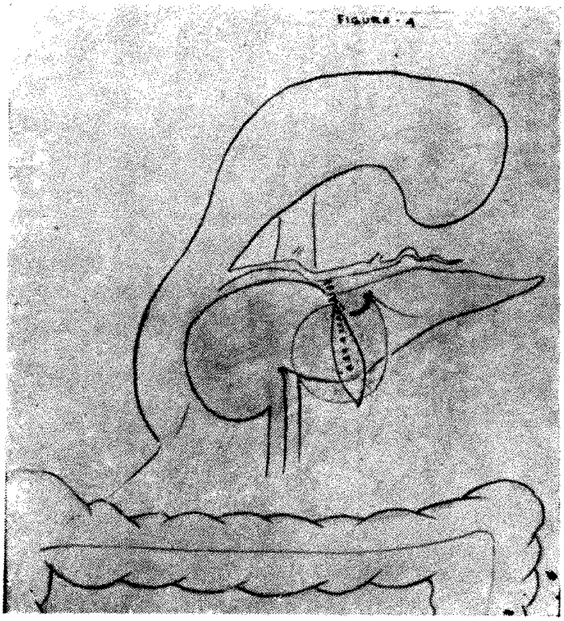

Fig. 4

Operative procedure

Operative procedures were on recommended lines but some of the special features adopted in our hospital I would like to emphasise :-

(a) Abdomen was opened through para median muscle splitting incision except in 3 cases. By rectus splitting approach abdomen could be entered within 3-5 minutes. Right or left, upper or lower depended on injury. Incision was extended further if needed.

(b) Operative examination is very important. Depending on the missile track, attention was focussed first on any bleeding sources which were either clamped, packed, ligated or sutured. After achievement of haemostasis attention was drawn to the large gut, urinary bladder, small gut, stomach, duodenum, liver, spleen, kidney in that order. Every part of the abdominal viscera was examined thoroughly to detect any hidden injury. Special care was given for the retroperitonial structures like pancreas, duodenum, duodeno-jejunal flexure, ascending and descending colon.

I like to give some of the illustrative examples which I had to tackle in our series:-

(a) **SW Back and Abdomen** - Shell piece entered through right loin, came out near the umbilicus. After opening the abdomen a tear on the anterior surface of ascending colon and 6 perforations of ilium were detected. The tear on anterior wall was ending laterally as if the missile entered through the lateral side. Apparently no other tear could be visualised. Only after mobilisation of the ascending colon another tear in posterior aspect, which was not connecting the anterior one, was detected. So mobilisation of the colon in this type of injury is very important (Fig. 1).

(b) **GSW Abdomen** – Wonud of entry was near the epigastrium, wound of exit was on the left side of lumbar spinous processess. On opening the abdomen a longitudinal tear in the antral region of stomach, a rent in the transverse mesocolon and inverted "V" shaped tear in duodenojejunal flexure were seen. After mobilising the duodeno-jejunal flexure and pulling out the duodenum from retroperitoneal space it was found that the tear was extending to the 3rd part of duodenum (Fig. 2).

(c) **GSW Abdomen** – Perforation of stomach, laceration through body of pancreas and laceration of left kidney. Stomach perforation was closed in two layers. Wouud of the pancreas was repaired with interupted silk, the peritonium covering the pancreas and leaf of transverse mesocolon was mobilised and brought over the pancreatic wound and sutured with continuous silk stitches. Nephrectomy was done through transperitoneal route and two drains, one intraperitoneal and another retroperitoneal were placed near the pancreatic injury. He was put on probanthine (Fig. 3 and 4).

(d) We had two cases of ascending colon injuries, in both the cases the tear was closed with two layers of silk without caecostomy. One case had an uneventful recovery, other case developed temporary faecal fistula but he started passing stool down below and faecal fistula closed spontaneously.

(e) **Rupture of Urethra and Bladder**–Conventional teaching of passing retrograde metal bougie did not work in my hands but passing the rubber catheter in a retrograde way and bringing the Foley's catheter per external urethral meatus into the bladder after tying tip to tip, worked very smoothly.

Post operative care

Routine post operative care was carried out. Reverine or penicillin and streptomycine were given to all as a prophylactic measure.

RESULT

6 cases made uneventful recovery and they were sent on sick leave.

5 cases were transferred to Army Hospital DELHI Cantt. Out of 5 cases, one to orthopaedic centre for fracture femur, one to reconstructive surgical centre for large wound on scapular region, one to Neuro surgical centre for sciatic nerve injury, one for Renal insufficiency and other for convalescence and expert care of pancreatic injury.

All were transferred after removal of sutures and they were on normal diet except one case who developed renal insufficiency.

Out of 16 cases mortality was only 5, which included 6 moribund cases. Thus overall survival rate was 70%. Out of 6 moribund cases two could be saved. If the moribund cases are excluded, out of 10 cases 9 survived. So the survival rate was 90%. Most of the moribund cases had multiple extensive visceral injuries including the blast effects on the viscera and nothing much in the form of surgery could be done for them.

CONCLUSION

16 cases of abdominal injuries during Indo-Pak war 1971 have been presented. Most of the patients had extensive multiple visceral injuries. Importance of mobilisation of colon and duodeno-jejunal flexure has been stressed upon for the detection of hidden injuries. Repair of right sided colonic injuries with two layers of silk without caecostomy was successful. Repair of pancreas with silk and retroperitonization resulted in healing without a fistula.

ACKNOWLEDGEMENT

I am deeply grateful to Lt Gen AK BARAT, PVSM, MVC, Chief Consultant in Surgery, for his encouragement and guidance in preparing this paper. I am also indebted to my Anaesthetist colleague, Major K RAMCHANDRA and paramedical staff for extending stupendous help and co-operation.

SUBACUTE SCLEROSING PANENCEPHALITIS

(Review Article)

BY

Major G.S. Octania*

INTRODUCTION

Subacute Sclerosing Panencephalitis (SSPE) is a rare disease which has come to limelight in the last six or seven years due to our better understanding of the slow virus infections. It affects the children of school going age and has an insidious and uneventful start. In due course, the parents note a behavioural disturbance in the affected child with progressive dementia. It is generally associated with epilepsy, myoclonic jerks, pyramidal and extrapyramidal signs. Sometimes there is akinetic mutism. The disease is rapidly progressive leading to death within a couple of months to two years. Spontaneous recovery, though rare has been recorded.

Nomenclature

The disease was recognised at the turn of the century by Von Bogaert as the subacute sclerosing leuco-encephalitis. In 1930's Dawson described it as 'inclusion body encephaltis'. A registry of .he disease was set up at the university of Tenesse in 1967 which accepted subacute sclerosing panencephalitis (SSPE) as the penultimate terminology.

Etiology

The etiology of SSPE has remained obscure until recently when the registry of the disease incriminated slow virus infection as the sole cause. Saunders et al. (1968) have postulated that immuno-competent lymphocytes specific for measles antigen may particularly be destructive to the host brain tissue leading to SSPE. Let us now consider the coutributions of the light microscopy, electron-microscopy and virology towards the elucidation of the viral etiology.

Light Microscopy. Viral etiology of SSPE was suggested by Dawson in 1933 due to the finding of Cowdrey type A intranuclear inclusions in the brain cells. Earlier workers thought these inclusions to be entirely due to Herpes Simplex virus. However, it is now known that measles-distemperrinderpest group of viruses, form similar inclusions. Conolly (1972) the chief virologist at the Royal Victorian Hospital, Belfast who is an authority on SSPE has described the disease in three boys aged 12, 15 and 17 years. These patients had childhood measles when they were 1, 3 and 4 years respectively. This meant there was a latent period of 11 to 13 years between the childhood measles and the onset of SSPE. Death occured in all the three cases between three to eighteen months. Following interesting points worth noting in these cases were :

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(a) None of the patients were related to each other or had any contact whatsoever.

(b) No history of terminal measles was forthcoming.

(c) No history of having received killed or live attenuated measles vaccine was available.

All the three cases reported by Conolly (loc cit) were confirmed histologically by the presence of intranuclear eosinophilic inclusions in the neurones and the glial cells. Methyl greepyronin technique stained the inclusions for ribonucleic acid, further indicating that a RNA virus such as measles was present. Feulgen stain was negative for DNA thus eliminating viruses like Herpes simplex, Herpes zoster and varicella.

Electronmicroscopy : Further support of the viral etiology of SSPE came from the electronmicroscopic studies of brain by Bourteille et al. (1965) and Tawara (1968). They found intrarucleat tubnlar filaments resembling those seen in dog kidney cells infected with measles virus. However, by electronmicroscopic studies alone it is impossible to identify any virus in particular since many closely resembling viruses have similar structure viz - measles, distemper, rinderpest, mumps, parainfluenza and other viruses of paramyxo group.

Virological techniques : First definite and direct proof of viral etiology was given by Conolly in 1968 in a number of cases of SSPE. In the cerebral eortex of these cases, he found measles specific fluorescence by direct immuno-fluorescence tests. Many attempts have been made to isolate measles virus from brains of affected cases but without success. Prior et al. (1968) have recorded a case of transmission of encephalitis from the brain biopsy material to the monkeys. Another team of workers have reported papova-like particles (tumour viruses) in the cultured human brain cells and presume that they are capable of causing disease with or without the help of measles virus.

Investigations

CSF shows normal cell count and pressure. The proteins are slightly elevated and the colloidal gold reaction shows a paretic curve. The electronencephalographic changes are not very conclusive and at times misleading when the record is abnormal on one side as compared to the other half, suggesting a space occupying lesion in the brain. The surest and the best way of diagnosis is by brain biopsy.

Immunology of SSPE

Measles induces both complement fixing and haemagglutinating antibodies in high titre in the sera of patients. CSF generally shows no change. In cases of SSPE however, the antibody titre is high both in sera and CSF. Conolly et al. (1968) carried out an interesting study about the immunoglobulins in measles and SSPE which are tabulated as follws :-

Immunoglobulin	10 cases of measles		3 cases of SSPE	
Level	Blood	CSF	Blood	CSF
IgG	++	—	++	++
IgM	++	—	++	++
Ig response	Disappeared by 46th day of illness		Persisted throughout the short course of illness	

It shows that IgG are probably produced within the CNS in cases of SSPE and that there is persistance of measles virus in this disease. Legg (1967) reported a case of SSPE who had spontaneous recovery. Periodical examination of serum from the case quoted above revealed marked fall in complement fixing antibodies but no change in the haemoglutinating antibody titre. Therefore, a fall in complement fixing antibody titre has prognostic value.

Pathological Lesions

Gross and microscopic lesions are only confined to central nervous system. All other organs reveal no remarkable pathology. Gross examination of the brain at autopsy shows marked atrophy and consequent widening of sulci. There are patches of discolouration and the consistancy of brain is firm. Microscopically, it requires some experience to interpret the slides lest they be confused with a mere case of encephalitis. Over and above the routine H&E staining, a battery of special stains is essential. They are Luxol fast blue for myelin fibres, Holzer's for glial fibrils and Landrum's phloxine tartrazine stain for inclusion bodies. Sections from the limbic area and brain stem reveal large number of intranuclear eosinophilic inclusions. Demyelination stands out prominantly everywhere. There is isomorphic gliosis and extensive mononuclear infiltration. There is remarkable fall out of neurones. The remaining neurones are few and far between and miserable looking with paucity of Nissl substance in them.

SSPE and Measles Vaccination

Measles vaccination has its own danger. Parker et al. (1971) reported six children who developed SSPE between three weeks to three years after immunisation with the live attenuated measles vaccine. It is therefore, essential that careful and prolonged surveillance be kept on children who receive vaccine since this rare complication may not appear until several years later.

SUMMARY

A complete review of the literature on SSPE till date has been carried out. There is a definite correlation between childhood measles and SSPE. Estimation of complement fixing and haemagglutinating antibody titre against measles in serum and CSF of patients is diagnostic of SSPE. The course of the disease can be tracked by periodical estimation of immunoglobulins. Proper attenuation of virus for vaccine production is of paramount importance and a watch must be kept for atleast two to four years after vaccination for any untoward complication.

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CLINICAL HAEMOGLOBINOMETERY

BY

Lt Col J.S. Paul*

INTRODUCTION

Haemoglobin values obtained by different methods currently used at Army Hospital, Delhi Cantt and their comparison with the values obtained by cyanmethaemoglobin method was the aim of this study.

Material and methods

Venous blood, taken in the morning between 9 AM and 10 AM, was collected in oxalate bottles from twenty patients. Haemoglobin estimation was carried out by the following methods :-

(a) Acid haematin (SAHLI's) Method.
(b) Oxyhaemoglobin method.
(c) Cynamethaemoglobin method.

Only standardised equipment and reagents were used in these methods. A brief description of oxyhaemoglobin and cynamethaemoglobin methods is given below.

Oxyhaemoglobin method - 0.05 ml of blood was taken in a blood pippette and delivered into a test tube containing 10 ml of 0.04 per cent, ammonia solution. The pippette was repeatedly washed in the solution which was then well mixed. The standard Lovibond comparator Disc 5/37 was used for taking the readings. The haemoglobin content could be read in the indicator window of the comparator by matching against the standards of the Disc.

Cyanmethaemoglobin method - Drabkin's solution was prepared by dissolving 200 mg $K_3 Fe (CN)_6$ (Potassium ferricyanide) and 50mg KCN (Potassium cyanide) in one litre of distilled water. The pH of the reagent was adjusted between — 7.0 to 7.4 and it was stored in a brown bottle which was kept out of direct light. Haemoglobin standard with cyanmethaemoglobin concentration of 61.6 mg per 100 ml was used (Biochemical unit, DELHI).

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0. 02 ml of well mixed whole blood was added to 4 ml of Drabkin's reagent. It was mixed by inversion and allowed to stand for at least ten minutes. Readings were taken on the spectrophotometer ("Spectronic 20"- Bausch and Lomb). Its wave length was at 540 mu. Calibration was done by taking first the readings of the cyanmethaemoglobin standard. Readings of the samples were then taken and calculation made according to the following formula:-

$$\text{Hb value G \%} = \frac{\text{Optical density of the sample}}{\text{Optical density of the standard}} \text{ X } \frac{\text{Concentration of the standard X Dilution factor}}{1000}$$

The data obtained are given at Table–I

Table

Values of Haemoglobin by different techniques carried out at Clinical Haematology Department, Army Hospital, Delhi Cantt

Method	Acid Haematin (SAHLI'S) Visual	Oxyhaemoglobin Lovibond's Comparator	Cyanmethaemoglobin Spectrophotometer (Spectronic 20)
Sr No.	Hb G %	Hb G %	Hb G %
1	11.4	12.0	13.1
2	13.2	15.2	15.4
3	8.4	11.3	11.2
4	12.8	14.8	15.0
5	11.0	13.5	13.9
6	12.0	15.8	16.2
7	11.0	16.0	16.2
8	8.6	9.5	9.7
9	14.6	17.0	17.9
10	16.2	17.8	18.7
11	11.4	12.5	14.6
12	11.0	12.0	12.6
13	13.2	14.0	14.6
14	13.8	14.5	14.6
15	14.0	14.6	14.9
16	14.6	16.0	15.9
17	14.2	15.0	16.0
18	14.8	16.0	16.8
19	15.4	16.5	16.8
20	15.6	17.0	17.1

DISCUSSION

The determination of haemoglobin is one of the most common tasks in the clinical laboratory and numerous methods have evolved over the past fifty years.

Advantages and disadvantages of various methods used are discussed below;

The necessity of adding water until a match is obtained, is a practical objection to SAHLI's method, as it is tedious and time consuming. It also takes long time to develop full colour of acid haematin. A truely satisfactory standard, which gives a perfect match has not also been devised, although a permanent glass *rod* is used as the standard.

In the oxyhaemoglobin method, the colour of an alkaline solution of blood is compared with a series of Lovibond permanent glass standards calibrated directly in grams of Hb per 100 ml of blood. The portion of haemoglobin that cannot readily be converted into oxyhaemoglobin is very small. The method is simple and minimum equipment is required.

The cyanmethaemoglobin procedure is the most widely used of all spectral methods and is the method recommended by the International Committee for Standardisation in Haematology in 1966. Oxyhaemoglobin, haemoglobin, methaemoglobin and carboxyhaemoglobin are converted to cyanmethaemoglobin. Sulphhaemoglobin is the only Hb pigment not measured by this method. Since this pigment cannot function as an oxygen carrier and is not reconvertible to haemoglobin it is relatively unimportant. The colour is stable for 48 hours. Cyanmethaemoglobin standard curve drawn from the figures obtained in our laboratory is given at Figure 1.

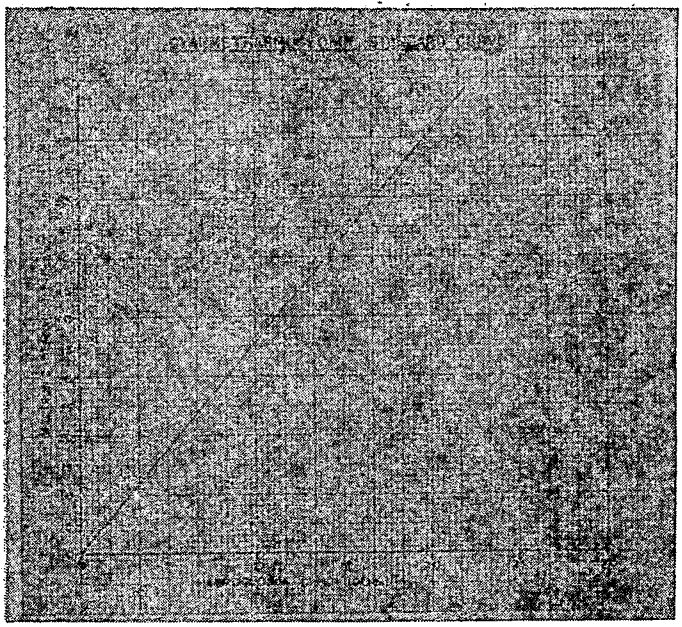

Fig. 1

The results show that lowest haemoglobin values were obtained by SAHLI's method. Oxyhaemoglobin method using Lovibond comparator gave results approximating those of the cyanmethaemoglobin method though slightly on the lower side. Spectral method gives precise and accurate readings. Cyanmethaemoglobin methed of haemoglobin estimation now internationally accepted and a large number of samples can be accurately studied in a short time. Its standard solution has also become available in the country recently. This method of haemoglobin estimation, should therefore be adopted in suitable service Hospitals.

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A NEW ENZYMATIC TEST FOR URINARY TRACT INFECTION

(A Preliminary Report)

BY

Lt Col M. R. Ray*

INTRODUCTION

Laboratory diagnosis of urinary tract infection still remains a problem. Symptomatology of the conditions is always not helpful for the diagnosis; moreover asymptomatic pyelonephritis can only be detected by proper laboratory aids like quantitative determination of significant bacteriuria. Asymptomatic bacteriuria of pregnancy needs early diagnosis so that further complications can be avoided. Even now the sheet anchor of the laboratory diagnosis of urinary tract infection is to determine the bacterial mass excreted out through urine. Kass (1960) suggested that 100,000 or more bacteria per ml of midstream urine is significant; 10,000 or less was to be considered as contamination from lower urinary tract and between 10,000/ml and 100,000/ml needed further investigation. Lampert (1971) suggested that 1000 organisms or more per minute in a timed specimen is significant and provides a better assesment than that suggested by Kass. Quantitative bacterial culture though desirable, in view of technical complexity, various screening tests like nitrite test, TTC test have been developed for the sake of simplicity and early diagnosis. It has been observed that these screening tests, which are based on the effects of the bacterial products of metabolism on certain chemicals are not very dependable when employed to determine the bacterial mass (Smith et al. 1961, Guze and Kalmanson 1963). It may be due to the variable behaviour of different strain of bacteria towards the chemicals.

Another great draw-back of all these tests including quantitative bacterial culture is, once an antibiotic is administered these tests are no more reliable.

Determination of the leucocytes excretion at times, provides additional data for diagnosis. Normal excretion rate of WBC, as reported in the literature are extremly variable; eg. 650,000 WBC and nonsquamous epithelial cell during 24 hours (Addis 1949); 28700 WBC/hour in male and 107,800 WBC/hour in female (Prescott 1966);

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180,000 – 196,000/litre (Hughten 1957). A short cut as suggested by Wing (1970) is to count the number of cells/cmm in uncentrifuged urine. If the number is more than 10/cmm the excretion rate alters due to conditions other than bacterial infection, e.g. in analgesic abuse and other non-infective pathological lesions. So the leucocytes excretion rate by itself is not considered a good screening test for urinary tract infection.

Serological rise in 'O' titre against the 'O' antigen of E.coli is more of value in confirming the type of E.coli infection and also probably of epidemiological interest.

A new test has been developed which is based on the alkaline phosphatase content of the urinary leucocytes (ULAP). The preliminary work suggests that it may provide a simple screening test for urinary tract infection and its progress during the course of treatment.

PRINCIPLE OF TEST

It has been observed that the leucocytes reacting to any inflammatory reaction contain more alkaline phosphatase than normal ones (Ray 1971). By cytochemical staining it can be demonstrated that urinary leucocytes voided in the urine during infection of the urinary tract contain more alkaline phosphatase than normal urinary leucocytes. Based on this fact quantitative ULAP has been determined and is found to be significantly high in urinary tract infection. The possible sources of the enzyme alkaline phosphatase in urinary deposits of physiological and pathological urine are (i) the excreted polymorpho unclear leucocytes (ii) exfoliated lining cells of proximal tubules (iii) some strains of bacteria (eg. E. coli). The method used for quantitative determination of the enzyme in urinary deposits produces complete lysis of the leucocytes but cannot lyse the bacteria. Exfoliation of the cells from the proximal tubules is generally not significant except in destructive lesions of these tubules. So, by and large AP of the urinary deposit is an expression of ULAP. Bacterial suspension of E.coli (Alkaline Phosphatase positive strain) does not show any enzyme activity by this method.

METHODS AND MATERIAL

Quantitative determination of ULAP is carried out in urinary deposits by using p-nitrophyenyl phosphate-as substrate and the result is expressed in International Units.

PROCEDURE

A sample of midstream urine is taken in a sterile widemouth test tube; the first part of the urine is collected in a urine glass for routine examination. A culture is put up and a careful routine examination of the urine is carried out.

15ml of the well mixed mid-stream urine is taken in a 70x10mm tube and centrifuged at 3000 rpm for 10mts. The supernatant is decanted off and the tube is inverted on a filter paper till the deposit is dry. Finally 0.1ml of distilled water is added to

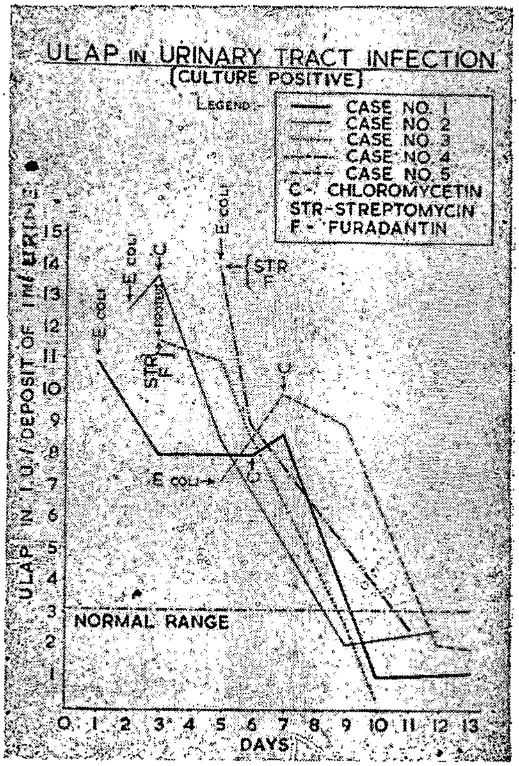
ULAP IN URINARY TRACT INFECTION
(CULTURE POSITIVE)
LEGEND:-
CASE NO. 1
CASE NO. 2
CASE NO. 3
CASE NO. 4
CASE NO. 5
C - CHLOROMYCETIN
STR - STREPTOMYCIN
F - FURADANTIN
ULAP IN I.U./DEPOSIT OF 1ml URINE
15
14
13
12
11
10
9
8
7
6
5
4
3
2
1
0
1
2
3
4
5
6
7
8
9
10
11
12
13
DAYS
E COLI
E COLI
C
STR
F
PROTEUS
E COLI
STR
F
C
C
E COLI
NORMAL RANGE

Fig. 1

suspend the deposit and the quantitative determination carried out by adding 0.5ml. of the substrate (Containing 2mg of p–nitrophenyl phosphate) and 0.5 ml of glycine buffer. EEL photo-electric colorimeter with blue filter OBIO (410 um) is used. For expressing in International units, the suspension of the deposit of 15 ml urine in 0.1ml distilled water is considered the test fluid and microgram of the substrate literated per minute per litre is indicated by International units.

To determine the base line of normal excretion, urine samples from 100 normal adult male and 100 adult non pregnant females were examined. 100 pregnant woman are also included in this series. In each case two different samples whether on the same day or on consecutive days were examined and the mean noted. Results of two different samples of each individual, though varied, were well within normal range except in two males where sperms were seen on microscopic examination these two were excluded. It is observed that there is no relation of ULAP to any specifie factor like time of collecting the sample, water restriction etc. First morning sample is not necessarily the most representative specimen. It is better to have a random sample in the forenoon. All these cases except four cases of pregnancy were culture negative and did not show any other abnormality.

Fifty cases of urinary tract infection of which 38 were culture positive and 12 clinically diagnosed but culture negative (some of them already had antibiotics before the specimen was sent to laboratory) were examined. A few cases of acute gluomerulonephritis, essential hypertension, severe diabetes, idiopathic albuminuria were also examined. The result of sequential study of five typical cases of culture positive urinary tract infection is presented in Fig. 1. It is observed that inital ULAP is much higher than the maximal normal range. The high level of ULAP persists till about 48 hours or more after administration of appropriate antibiotic and then it shoots down to normal pari passu with complete relief of symptoms. The twelve cases clinically diagnosed as urinary tract infection but culture negative also showed initial very high ULAP which came to normal after treatment except in two cases who continued to have very high ULAP in spite of treatment with furadantin, sulphamezathin and finally chloromycetin. In these two cases there was no relief of symptoms either. These two cases showed high ESR with positive Mantoux test but without any radiological evidence of tuberculosis in lungs. Follow up of these two cases was not possible.

Sequential study of four cases of acute glomerulonephritis, two cases of idiopathic albuminuria, four cases of hypertension (essential) five cases of severe diabetes did not reveal any remarkable rise of ULAP beyond high normal range.

RESULTS

Table 1

ULAP in 100 Healthy/Young Soldiers

Ser No.	ULAP in deposit of 15 ml (I.U.)	No. of Cases
1	5—15	70
2	16—25	15
3	26—35	7
4	36—45	8
Range — 5-45 I. U.	Total	100

Table 2

ULAP in 100 Non - Pregnant Adult Women
(Medical students, Nursing Students, Nursing Officers)

Ser No.	ULAP in deposit of 15 ml (I. U.)	No. of cases
1	5—15	60
2	16—25	30
3	26—35	8
4	36—50	2
Range 5 — 50 I. U.	Total	100

Table 3

ULAP in 100 Pregnant Women in Different Phases of Gestation

Ser No.	ULAP in depost of 15 ml (I. U.)	No. of Cases
1	5—15	24
2	16—25	10
3	26—35	20
4	36—45	20
5	46—55	12
6	56—65	4
7	Above 65	10*

*Out of these 10 cases 8 cases were put up for culture. Six of these eight cases were culture positive.

Table 4

ULAP in Different Renal Conditions

Diagnosis	ULAP in deposits of	No. of Cases
Normal adults male	3—45	100
Normal adults female	5—50	100
Normal pregnancy	5—65	100
Urinary tract infection	90—400	50
Acute glomerulonephritis	30—48	4
Essential Hypertension	30—45	4
Severe Diabetes	10—30	5
Albuminuria (idiopathic)	10—28	2

DISCUSSION

The ULAP estimation for clinical diagnosis is a new attempt. The preliminary study of 50 cases of urinary tract infection shows a marked elevation of the ULAP which persists for 48 hours or more even after the administration of appropriate antibiotic. The reason for such elevation is increased enzyme content of the urinary leucocytes reacting to inflammation. Sequential study of acute glomerulonephritis, essential hypertension, idiopathic albuminuria, severe diabetes did not reveal any remarkable rise of ULAP. The method is very simple, reproducible and does not require any aseptic procedure. Detailed studies of a large number of cases of various other pathological conditions as well as urinary tract infection are being carried out under the Armed Forces Medical Research Committee Project.

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MEDICOLEGAL AUTOPSY

BY

Major Ajit Singh*

INTRODUCTION

A medicolegal autopsy is carried out to assist the authorities in the administration of justice as compared to a pathological autopsy which is primarily performed to determine the cause of death and to correlate the clinial findings with the existing pathology in the body at the time of death.

There will be certain occasions where the cause of death may be clearly established by relatively inexperienced examiner but generally specialised under the performance of a medicolegal postmortem examination is a highly specialised under taking requiring skill and experience. This becomes all the more important when it is realised that the findings of a medicolegal autopsy may later be the subject of close legal scrutiny.

Many Medical Officers may never have occasion to perform a postmortem examination although the authorities concerned can direct any Registered Medical Practitioner to perform ar autopsy. Time and again Service Medical Officers (Pathologists in particular) have found themselves in a difficult position regarding the medicolegal autopsies as many details are not covered by the lectures given to them for their training in the Armed Forces at various stages. This article incorporates such details and is intended to solve some of their problems on the subject.

OBJECTS OF A MEDICOLEGAL AUTOPSY

The main objects of performing a medicolegal postmortem examination are :–

(a) To establish the identity of the dead body when it is not known. It is the responsibility of the Police Authorities to arrange for the photographs and finger prints to be taken by the respective experts. A Medical Officer should not perform an autopsy on an unknown body unless these formalities have been fulfilled or the body properly identified.

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(b) To determine the time since death.

(c) To find out whether the individual has died of a natural or an unnatural cause.

(d) To find out the nature of death, whether it is suicidal, accidental or homicidal.

(e) In newly born infants to determine :-

(i) Whether the infant was born alive or dead

(ii) If born dead, whether it was viable or not.

It is not always possible to arrive at the exact conclusions in every case but the medical officer should perform the autopsy carefully and note down the various details accurately in a systematic manner. An excellent account of medicolegal autopsy technique is given by Glaister and Rentoul (1966) and the relevant rules on the subject are described by Modi (1969). It is always better to read such manuscripts before performing a medicolegal postmortem examation.

AUTHORITY : WHO CAN ORDER A MEDICOLEGAL AUTOPSY

A person who has been authorised by law to hold an inquest in cases of unnatural deaths or deaths occuring under suspicious circumstances can order performance of a medicolegal postmortem. In accordance with the provisions of Section 174, Criminal Procedure Code, the responsibility for holding an inquest devolves on the local civil authorities. The following persons are authorised to order a medicolegal autopsy.

1 CORONER in the cities of Bombay and Calcutta.

2 MAGISTRATE of the area concerned at all other places in India. Usually this power is delegated to the police officer incharge of the local police station.

3 PRESIDING OFFICER of a Military Court of Inquest in places where such an inquest is being held.

4 FORMATION COMMANDER in operational areas where civil authorities do not have facilities to arrange a postmortem examination and may request a Formation Commader to arrange the same in a Military Hospital (AO 484/68 refers).

It is therefore quite clear that an Officer Commanding a Military Hospital has no authority to order a medicolegal postmortem examination. Written information regarding cases of unnatural or supicious deaths of persons subject to the Army Act or of those not subject to the Army Act which occur within the Unit Lines will be conveyed by OC Unit immediately on occurence, BY HAND, to the local civil police officer. Where the civil authorities are not easily accessible as in operational areas a general approval for conducting Military Courts of Inquest should be obtained in advance from the State Government concerned.

PERSONS AUTHORISED TO PERFORM A MEDICOLEGAL AUTOPSY

A postmortem examination is not essential in every case of unnatural death although an inquest is a must. When postmortem is decided to be carried out by the concerned authorities, they may have the dead body sent to the nearest CIVIL SURGEON or other qualified medical officer appointed in this behalf by the State Government. A Service Pathologist who has not been so appointed, CANNOT, therefore be compelled by the Civil Authorities to carry out a medicolegal autopsy whether outside or inside an operational area.

A Service Medical Officer can, however, be ordered to perform a postmortem examination by an appropriate Military Authority when a Formation Commander in an operational area decides to accept a request from the Civil Authorities to arrange the same in a Military Hospital.

When a Military Court of Inquest is held, a postmortem examination may be carried out by Military Medical Officers in cases where such examination is necessary.

Sometimes a Service Pathologist is asked to perform a medicolegal autopsy on the basis of a NO OBJECTION Certificate obtained from the police by the OC Unit/Hospital. This is not only an irregular practice but also illegal. Service Medical Officers should carry out the medicolegal postmortem examination only when an order/request is received from the Formation Commander under the circumstances already explained.

Detailed instructions on this subject are given in AO 484/68

UNNATURAL OR SUSPICIOUS DEATHS

The following types of deaths come under this category and an inquest must be held in such cases :–

(a) All unexpected/sudden deaths.

(b) All cases of murder or suicidal deaths.

(c) All deaths by accidents or poisons.

(d) All cases of infanticide.

(e) All deaths during surgical operations.

(f) All deaths due to anaesthesia.

(g) Deaths of a convict in Jail.

(h) Deaths of a person in police custody.

(j) Death of a patient in a mental hospital.

(k) All cases where the cause of death has not been certified by the attending medical officer and there is a suspicion of foul play.

SOME USEFUL HINTS FOR SERVICE PATHOLOGISTS

(a) Satisfy yourself that the order/request for a medicolegal postmortem has been received from the Appropriate Authority. It is your duty to bring it to the notice of the OC Military Hospital if an irregularity is being committed.

(b) Study the case notes, inquest report and other available documents before performing an autopsy. A careful history obtained from the relatives and friends is always helpful in reaching your conclusions.

(c) It is alawys better to go through the various regulations and follow the correct procedure to aviod any unnecssary enquiries later on.

(d) Do not commit anything to your memory. Make all the notes carefully and in detail in a systematic manner at the time when autopsy is being done.

(e) If your findings are inconclusive, you must preserve the appropriate material for histopathological, biochemical and bacteriological examination.

(f) In all cases of POISONING, you must collect, preserve and despatch the appropriate visceras and specimens for chemical analysis.

(g) If you come across any evidence suggesting unnatural or suspicious death at any stage during the performance of a pathological autopsy, it is your duty to close the autopsy at that stage and inform the authorities concerned in WRITING about your findings.

(h) When in doubt, consult a senior colleague.

(j) Lastly remember, that your duty is only to assist auhorities in the administration of justice and not to sit at the judgement yourself.

SUMMARY

Some current rules and regulations dealing with the medicolegal autopsies have been discussed for the benefit of the Service Medical Officers. These should be strictly complied with to avoid any embarrasement to all concerned in a court of law later on.

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1 Army Order 484/68

2 Criminal Procedure Code – Section 174

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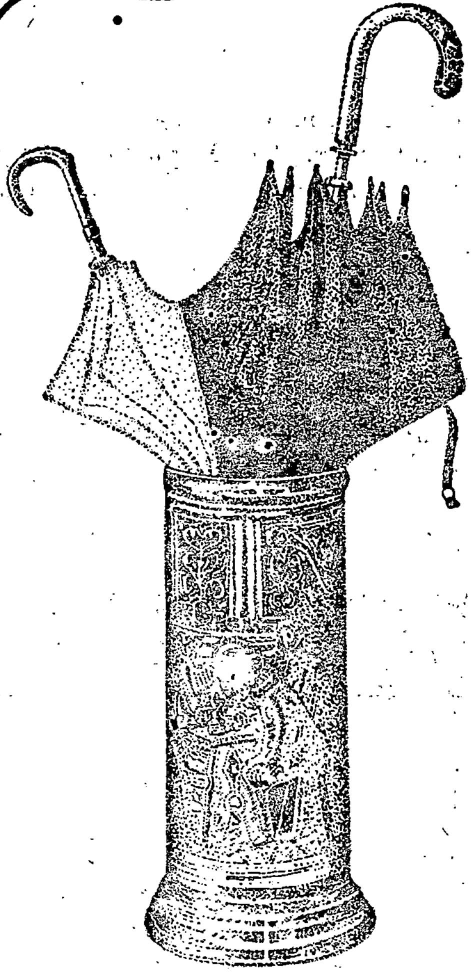

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INCIDENCE OF AUSTRALIA ANTIGEN AT HIGH ALTITUDE

BY

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Australia antigen, now known as hepatitis associated Australia antigen (HAA) first described by Blumberg (1964) has been increasingly associated with viral hepatitis and cirrhosis (Blumberg et al, 1967a, Prince 1968, Okochi and Murakami, 1968; Bayer et al. 1968; Wright et al. 1969; Sherlock et al. 1970; Sehgal and Aiket, 1970; Sama et al. 1971; Bagshawe et al. 1971) and other hepatic diseases including hepatic amoebiasis and primary liver cancer. Among the non-hepatic groups of diseases, the antigen has been found to be associated with Down's Syndrome (Sutnick et al., 1967), leukaemia, lymphoma and lepromatous leprosy (Blumberg et al. 1965; Blumberg et al. 1967a, Sehgal and Aikat,1970). Sehgal and Aikat, (1970) have also mentioned the presence of HAA in isolated cases of nephrotic syndrome, multiple myeloma, S.L.E., rheumatoid arthritis, Takayasu disease, pulmonary eosinophilia and paraplegia. Whether there is an association between HAA and diseases is still to be elucidated.

The nature of the infective agent responsible for HAA positive liver diseases is largely unknown, but the recent work of Dane and associates (1969) has shown that 42 nm. Dane particles are the actual virus and the virus consists of an outer coat of HAA and inner spherical 27 nm components resembling a rhinovirus (Almeida et al., 1971). However, HAA has been shown to contain multiple antigenic components (Le Beuvier, 1971). It has been convincingly proved that the infective agents of infective hepatitis and serum hepatitis can be transmitted by oral route (Krugman et al. 1967). Prince feels they are immunologically identical or atleast share a common antigenic site (Prince 1969).

Contact with material containing HAA may result in no apparent effect or in hepatitis of varying severity (Dudley et al. 1972). It is known that severity of hepatitis once acquired at high altitude is rapidly progressive (Singh, 1972). Therefore, it was thought that the incidence of HAA at high altitude might provide some information of relevance. Reports on the incidence of HAA in India vary (Sehgal and Aikat, 1970; Sama et al., 1971). However, several workers are currently assessing the incidence in selected Indian population (Chandra, 1971; Malaviya, 1971). It was felt that since the incidence of HAA at high altitude in India was not known and in view of the reported high incidence in the Western high lands of New Guinea, perhaps HAA occurrence would be different in high altitude residents in the Himalayan ranges.

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MATERIAL AND METHODS

Four hundred persons normally resident in the plains (Delhi, 650 feet) were tested and designated the control population. One hundred and fifty high-altitude residents (high-altitude natives) were included in the study. Sera of high altitude adapted individuals resident at the altitudes varying from 12,500 feet to 18,000 feet for 2 years were collected at varying times and subsequently 350 sera were tested. Standard antigen (Hepatitis associted antigen) and antibody were supplied by the courtesy of Dr. H.G. J. Brummelhuis of the Central Laboratory of the Netherland Red Cross Blood Transfusion Service, Amsterdam. The micro-Ouchterlony technique was routinely used and subsequently electro-osmo diffusion (E.O.D.) was employed. Standard microscope slides 3″ × 1″ were coated with a 6 mm layer of 0.9% agarose gel in buffer (Tris, 0.01 M, pH 7.6, saline solution 0.1 M and EDTA, 0.001 M containing 1 mg/ml protamine sulphate). This method would eliminate non-specific reactions between adjacent wells. A standard punch was used to cut 6 wells 2 mm in diameter around a central well. The Peripheral wells were 3 mm from the central well. Test sera for determination of HAA were placed in the peripheral wells and standard antiserum was placed in the central well. Incubated was also a control HAA. The plates were incubated in a humid chamber at room temperature and were observed at 24 hours' intervals for a week (Prince, 1968).

Electro-osmodiffusion was carried out in a concentration of ionagar 1.5% in veronal buffer at pH 8.6. Precoated frames with 2 mm thickness of agar were used. Wells 3 mm in diameter, 7 mm apart were cut. 10 micro-litres of Australia antiserum were added to the well near the anode end and an equivalent volume of patient's serum in the well near the cathode end was applied. Electrophoresis was carried out for two hours at a constant voltage of 7 to 8 volts per cm and 20 mili Amps per immunoframe. Presence of Australia antigen was indicated by the precipitation lines between the wells (Yap et al. 1971). Quantitative measurements of immunoglobulin IgG, IgA and IgM were carried out in 31 sea level subjects, 10 healthy high-altitude natives, and 4 HAA positive high-altitude natives, by radial diffusion method of Mancini as modified by Fahey and McKelvey (1965). Immunoglobulin standards were supplied by the courtesy of Drs. John Fahey and D. Rowe, WHO Ref. Laboratory (No. 67/97).

RESULTS

Results obtained were compared with those of other populations tested by similar methods. Table 1 shows the comparative values obtained. The values for Chinese, Malay and Malay Indians are those of Dr. Malcolm Simons of WHO Immunology Centre, Singapore (Simons, 1971). The figures obtained on Indian Population were not significantly different, however, the high-altitude natives showed a significantly higher incidence of HAA.

Table 1

Comparison of Incidence of Australia Antigen in Different Ethnic Types

Ethnic Type	Number tested	Number positive	Percentage positive
Chinese (Simons, 1971)	1632	69	4.2
Malay (Simons, 1971)	445	7	1.6
Indian (Malay) (Simons, 1971)	248	2	0.8
Indian (India) (Present study)	400	2	0.5
High-altitude natives (Present study)	150	4	2.7

Table 2 shows comparative values obtained in sea level residents, high-altitude adapted residents and high-altitude natives. There was no significant difference in the high-altitude adapted individuals and persons resident at sea level. However, the high-altitude permanent residents (natives) showed an increased incidence (2.7% $P<0.001$). Using E.O.D., one more positive reaction was obtained in sea level individuals and high-altitude adapted persons. This did not materially alter the significant high level of incidence in high-altitude natives.

Table 2

Comparison of incidence of Australia Antigen in Sea Level subjects, high-altitude adapted individuals and high-altitude natives

Category	Number tested	Number positive	Percentage positive	Significance
Sea level subjects	400	2	0.5	Not significant
High-altitude adapted individuals (over 2 years)	350	1	0.3	$P<0.001$
High-altitude natives	150	4	2.7	

Table 3 shows the comparative values of immunoglobulins in sea level subjects, HAA negative high-altitude natives and HAA positive high-altitude natives. It can be seen that IgG and IgA are significantly higher in HAA-nagative high-altitude natives and IgM values though numerically higher are not significantly different when compared with sea level values.

Table 3

Comparison of immunglobulins levels in sea level subjects, H.A.A.-negative high-altitude natives and H.A.A.-positive high-altitude natives

Category	Immunoglobulins mg % serum IgG Mean ± SD	 IgA Mean ± SD	 IgM Mean ± SD
A. Sea level subjects (31)	1139 ± 254	205 ± 49	156 ± 50
B. HAA-negative high-altitude natives (10)	1742 ± 515	288 ± 37	184 ± 65
C. HAA-positive high-altitude natives (4)	2404 ± 363	393 ± 141	196 ± 107
Significance	B v/s A **	**	NS
	C v/s A **	**	NS
	C v/s B *	*	NS

Figures in parenthesis show number of persons tested.

**Significant at 1% level * Significant at 5% level NS Not Significant

Although HAA positive high-altitude natives show a similar trend, the values of IgG and IgA showed a significant increase even above those of HAA negative high-altitude natives. Therefore, it would seem that these immunoglobulins are raised when concomitant persistence of HAA is present. The individual levels of immunoglobulins in four HAA positive high-altitude natives are shown in Table 4.

Table 4

Immunogolbulin levels in 4 H.A.A.-positive high-altitude natives

Srl No	Immunoglobulins mg % serum IgG	 IgA	 IgM
1	2790	338	351
2	1976	567	109
3	2600	235	192
4	2250	432	172
Mean	2404	393	196
SD	±363	±141	±107

DISCUSSION

The persistence of HAA in high-altitude natives seemed intriguing as consistently lower levels have been reported in Indian population, ranging from 0.4 % (Sama et al., 1971) to 0.7% (Chandra, 1971) using immunodiffusion technique. Whether genetic factors are important in determining the persistence of HAA in various ethnic groups is worthy of consideration (Blumberg et al., 1970). It would appear that the severity of the disease and persistence of the antigen would be determined by environmental factors, genetic factors and acquired variations in immnue response.

Environmentally, the high-altitude natives live in crowded accommodation with minimum of sanitary facilities. This could contribute to the persistence of a nidus of infection in the community. Ethnically, the populations rarely interbreed with others. It is konwn that the incidence of Australia antigen is high in populations such as, Indonesians, 5.9%, New Guinean high-landers 13.6% and Chinese 7.5 (Simons, 1972). It is interesting to note that highest incidence is in the highlands of New Guinea. Whether the altitude per se or due to its detrimental influence on the immune response plays a part in the persistence of the antigen is speculative at present. It is known that HAA is likely to persist after subclinical hepatitis and is possibly associated with an impaired T-cells reponse (Barker and Murray, 1971; Dudley et al., 1972) as demonstrated by a poor response to dinitrochlorobenzene (DNCB) (Dudley and Sherlock, 1972). It is also possible that persistence of the antigen in turn could in some measure contribute to the chronically raised immunoglobulins levels found in natives at high altitude (Table 3). This evidence in HAA positive high-altitube natives in whom higher levels of immunoglobulins are present, concomitantly with persistent antigenaemia (Table 4). Persistence of HAA is encountered in conditions with impaired cellular immunity like lepromatous leprosy (Blumberg et al., 1968), leukaemias and lymphomas (Blumberg et al., 1967a) and Down's Syndrome (Blumberg et al., 1967b). In several instances, such as, leprosy (Turk and Bryceson, 1971) tuberculosis, Hodgkin's disease (Aisenberg, 1962; Brown et al., 1967), myeloma and leishmaniasis (Convit, 1970; Turk and Bryceson 1971). There is a diachotomy between humoral and cell-mediated immunity (Asherson, 1966; Burnet, 1968). Increased immunoglobulin levels with decreased response to phytohemagglutinin (PHA) have been notable features in the above situations. It is interesting to note that tuberculosis where T-cells responses are thought to be impaired, also is prevalent in natives at high altitude, although humoral responses appear to be intact or even augmented. Clinical experience shows that tuderculosis, leprosy, pneumonia, amoebic and viral hepatitis follow a fulminant course at high altitude despite increased immunoglobulin levels at high altitude. It is possible hypoxia of high altitude could adversely affect the cell mediated immunity and it would be pertinent at this juncture to enquire into the status of cell mediated immunity in residents at high altitude especially so as Prof Sherlock and Dudley have hypothesised an impaired CMI response being contributory in hepatitis (Dudley and Sherlock, 1972).

SUMMARY

Four hundred sea level subjects, 350 high-altitude adapted individuals and 150 high-altitude natives were tested for Australia antigen by both micro-Ouchterlony and electro-osmodiffusion techniques. The incidence of HAA was found to be significantly higher in high-altitude natives whereas no significant difference was found between sea level subjects and individuals adapted to high altitude. The possible reasons for the higher incidence and persistence of the antigen in high-altitude residents have been discussed in this report for the first time in the Himalayas.

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MEDICAL COVER DURING OPERATION IN HIGH ALTITUDE AREA AND EXTREME COLD CLIMATE

BY

Lt Col S. N. Aich*

INTRODUCTION

221 Medical Company had the unique experienee of providing medical cover to an Infantry Brigade Group located in high altitude area during Indo-Pak war of December 1971. The formation therefore fought against three enemies simultaneously. The first of-course was the Pakistanis, the Second High Altitude and the third Sub-Zero temperature,

AIM

While providing Medical Cover to the formation during the war, the medical company therefore came across with many problems peculiar to the terrain and climate. This article intends to describe the problems, how these were solved and the experience gathered so that in case of future operations under similar conditions the medical planning is not handicapped.

PROBLEMS

Effects of Extreme Cold Climate.

Frost Bite :–The atmospheric temperature during the period of war was between maximum + 1°C to + 5°C to minimum – 20°C to –25°C. The operation was usually fought during the night and hence the troops were always exposed to minimum temperature. Though all precautions.. as far as possible under the operational conditions, against the effects of cold were taken by the troops, yet the incidence of frost–bite cases was collosal. Complete ECC clothing was used by the troops. Most of the battalions suffered Frost-bite casualties during the offensive role when they were in the open. The incidence of frost bite cases was much reduced when the troops could move into some sorts of shelter left by enemy. Over head shelter, therefore is an important factor for prevention of the effects of cold. Wind velocity at that time used to be from 0.5 to 28 knots/hour. Overhead shelter therefore provided protection against the effects of cold by reducing the wind chill factor.

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In an offensive battle in Sub-Zero temperature where troops will hardly have any opportunity to remain under shelter, certain wastage of man power due to Frost-bite will have to be accepted. In the experience of this Medical Company 3 to 6% of the strength of the Infantry Battalion should be always expected as casualties due to effects of cold during the first few days of an offensive operation. This percentage of casualties should be suitably varied depending upon how long troops remain exposed to Sub-Zero temperature in the open.

Drinking Water :-Drinking water for the assaulting troops was another problem in the extreme cold climate. Most of the streams were frozen. Though troops carried water in water bottles, yet it was of no use, as the water in the water bottles used to freeze. In most of the time they quenched their thirst by swallowing softs now. Some lit fire if situation permitted, to melt the frozen water in water bottle. One officer claimed that he added some Rum in the water bottle and his bottle was not frozen. Alcohol has low freezing point but it is doubtful whether addition of alcohol to the water bottle to the extent of not producing any adverse effect to an average soldier, could prevent freezing of water. This problem requires to be looked into and some additive should be thought of to prevent freezing of water in water bottle, so that troops do not face this difficulty again if they are to operate in Sub-Zero temperature.

Hospital :—In functioning of the hospital, the extreme cold proved to be both advantage and disadvantage. The only advantage was that though the Medical Company did not have any storage facilities for blood but due to extreme cold and availibility of frozen water, storage of blood did not pose any problem. The main disadvantage was to keep the hospital wards and departments warm under operational oonditions, by using oil-Bukharies. Due to continuous subzero temperature the hospital faced a peculiar problem. It was found that all the injections and transfusion fluids were frozen and their glass containers were cracked. Promptly a small room, properly heated by burning an oil-Bukhary continuously was earmarked for storage of all transfusion fluids and injectable. There-after no dfficulty was faced. But some times it was observed that while transfusion was going on to the patient in wards, some icecles appeared in the bottles particularly those containing Mannitol. Frequent shaking of bottles prevented formation of icecles. Therefore, not only the wards should be heated but also the transfusion fluids and the injectables should be stored in a heated room to prevent wastage due to freezing and cracking of the containers.

Operation Theatre :-The operation theatre at OP location was heated by oil-Bukharies. But before any anaesthetic was used or any case was brought in for operation, the Bukharies were put off so that there was no naked fire in the theatre. This worked all right but in some cases where an operation took more than 3-4 hours, the operation theatre used to get cold, much to the inconvenience of the theatre-staff.

Effects of High Altitude :—Conversely casuality due to effects of high altitude was Nil, though the troops operated in high mountains of altitude varying from 12000 to 16000 feet. Low incidence of effects of high altitude during winter is a known factor. The plausible reasons for the same is that normally during winter high mountain posts are vacated due to snow and movements of troops in the form of long range patrols are restricted. But none of these reasons could be attributed to the strikingly low incidenee of effects of High Altitude in this particular case where troops operated in high altitude in temperatures varying from +1°C to −25°C. What is the cause of such low incidence of effects of high altitude during extremely cold climate? Does the extreme cold climate increase the tolerance of an individual to the effects of hypoxia or it is just a peculiar finding unconnected with any factor?

Evacuation of Casualty from forward Area :—Evacuation of casualties from forward areas to the base in the high mountainous area was a challenging problem. War in mountatin was fought in many areas simultaneously and hence the routes of evacuation of casualtles were multiple. In most of the cases there was no track even and evacuation was done through cross-country routes. The capabilities of the stretcher-bearers were limited due to high mountains, extreme cold climate and long route of evacuation. The earliest time in which a casualty was brought to base by the SB's for evacuation by Ambulance Car was eight hours and the latest upto 48 hours. It was not possible to establish SB Relay Posts enroute as the routes of evacuation from forward areas were multiple and varied. Besides it was not advisable to put a couple of SB's enroute all by themselves. endangering their personal safety. Casualties in most of the cases were evacuated by one SB squad, taking rest enroute as many times as they required.

It is beyond the capacity of any field medical units with its present organisation to take the complete responsibility of evacuation of casualties by SB's in the high mountainous area. Casualty evacuation and logistic support to forward troops posed similar problems. The logistic support was maintained by mustering adm personnel of the respective units and civilian porters. The same people were used to evacuate casualties while returning to the base. If logistic support and casualty evacuation were not planned and executed simultaneously, it would not have been possible for the Medical Company to evacuate as many as 710 casualties including frost-bite cases during the war. Evacuation of casualties from forward areas by SB's is tedious, time consuming and most uncomfortable to the patients but is dependable. Experience of this Medical Company is that while planning casualty evacuation in high mountainous area, it has to be done jointly with logistic support in close liaison with the staff of formation headquarters.

Due to long route of evacuation of casualties by SB's four-hourly staging could not be done. The casualties reached the Surgeon after 8 to 48 hours of sustaining the injury in forward area. But surprisingly the incidence of wound infection was nil and this is probably due to extremely cold climate. Hence in extreme cold climate if a casualty does not reach Surgeon within 6 hours, not much harm is caused because of the absence of wound infection.

Air Evacuation of Casualty :-Evacuation of casualties by helicopters from forward areas is ideal but not practically possible due to many limiting factors. First is the availibility of helicopters and next is the availibility of helipads, particularly in the enemy territory. Construction of helipads in the rocks of high mountains within a short time is impossible. Even if both helicopters and helipads are available, air evacuation cannot be done because of the enemy artillery firing over or near the landing place. This medical company could not evacuate casualties by helicopter for first two days of operations from existing helipads due to enemy fire.

Evacuation of Casualty to Base Hospital :-The casualties from hospital of the Medical Company were evacuated to base hospitals located at distance of about 150 miles. Air evacuation of casualties to base hospitals could not be done due to non-availibility of transport aircraft. However serious casualties were evacuated to Base Hospitals by helicopters. Priority II & III cases were evacuated by road which took nearly 12 hours enroute one way. As the Ambulance Cars held by the unit were used for evacuation of casualties from ADS and Ad-hoc Medical Aid posts to the hospital, stretcher fitted 3 Ton GS vehicles were used for evacuation of casualties to Base Hospitals. Four stretcher beams were fitted in each GS Vehicle and hence each of them could carry 4 lying and 8 sitting cases or 16 sitting cases only. The vehicles were useful because of their increased carrying capacity and long route of evacuation but were very uncomfortable for the patients due to less protection against cold enroute.

CONCLUSION

Evacuation of casualties in high mountainous area remains an ever-challenging problem. Air evacuation from forward posts is ideal provided it is practicable and if the OP situation permits. Evacuation of casualties by SBs/civilian porters is rather dependable but involves lot of problems and therefore it is not ideal. A combination of these two modes of evacuation of casualties from forward areas may be the answer and hence it is worth-trying in future operations under similar condition.

NEONATAL JAUNDICE ASSOCIATED WITH HETEROSPECIFIC PREGNANCY

BY

Major B. S. Chakrabortty*

The occurrence of jaundice in newborn is well recognised and is responsible for high rate of mortality and morbidity. The incidence of clinical jaundice has been recorded as high as 54.6% in a total of 1281 live births studied by Bajpai et al (1971). The authors reported 18% of cases originating as a result of ABO and Rh blood group incompatibilty, which remained as the commonest known etiology as seen from Table I.

Table I

Etiological classification of neonatal Hyperbilirubinaemia (Bajpai et al – 1971)

Srl No	Etiological factor		Percentage of Cases
1.	ABO incompatibility		16
2.	Rh incompatibility		2
3.	Sepsis		14
4.	G–6–PD Deficiency		4
5.	Miscellaneous causes:	(a) Cephlahaematoma	4
		(b) Anoxia	4
		(c) Hypoglycaemia	2
		(d) Excessive Vit K administration	2
6.	Physiological jaundice with prematurity		14
7.	Unknown		38

Maternofetal blood group incompatibility is the result of heterospecific pregnancy. Hirzfeld (quoted by Mollison, 1967) originally applied the term 'heterospecific pregnancy' to all pregnancies in which blood groups of mother and infant were different, but it is now restricted to cases in which the red cells of the foetus contain an antigen derived from its father, which is foreign to the mother. The onset of jaundice in neonates at birth and within 24 hrs of birth should always suggest haemolytic process (Mollison, 1967). The purpose of this communication is to emphasize the occurence of neonatal jaundice in newborn babies within 24 hours of birth which is associated with heterospecific pregnancies in multiparous women.

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MATERIALS AND METHODS

Twenty cases of neonatal jaundice were studied. Blood from these babies alongwith blood from both of their parents were received in Blood Transfusion Department, Armed Forces Medical College, PUNE (Maharashtra) during the period from 24 April 1971 to 09 April 1972. All the newborn babies developed jaundice within 24 hrs of birth. Blood from these cases were sent for further tests to confirm the clinical suspicions of haemolytic disease of newborn. These were based on the onset of jaundice and the blood groups of parents of these babies. The following investigations were carried out from the blood samples :—

1. Tests carried out on mothers' blood :—

(a) ABO and Rho (D) grouping.

(b) Tests for Rho (D) antibodies and determination of antibody titre were carried by methods of saline agglutination at 20°C, albumin agglutination at 37°C, enzyme agglutination at 37 C, and indirect antihuman globulin method at 37°C.

(c) Detection of Immune anti-A and anti-B and determination of titre were carried out by using indirect antiglobulin test following neutralisation of saline agglutinin by group specific substances (Dunsford and Bowley, 1967).

2 Tests carried out on fathers' blood :—

ABO and Rho (D) grouping.

3 Tests on babies' blood :—

(a) ABO and Rho (D) grouping.

(b) Direct antiglobulin tests.

RESULTS

The study on selected 20 cases of neonatal jaundice associated with heterospecific pregnancy yielded only 11 group 'O' mothers; 7 of them had group 'A' children, out of which only 2 cases showed evidence of isoimmunisation in mothers. 4 group 'O' mothers had children of group 'B' and none of them showed evidence of isoimmunisation. There were 9 Rho (D) negative mothers (Group 0=4 and non group 0=5) who had Rho (D) positive children. Only two mothers showed evidence of isoimmunisation (1 in group 0 and 1 in non group 0 mother). The results are summarised in Table II.

Table II

Showing ABO and Rho (D) blood group incompatibility and isoimmunisation in 20 cases of neonatal jaundice.

Cases showing incompatibility			Cases showing isoimmunisation	
No. of mothers	Children			
	Number	Percentage out of 20	Number	Percentage
Group 0=11	Group A–7 Group B–4	55	Group A–2 Group B–0	18 (out of 11)
Rho (D) negative 9	Rho (D) positive 9	45	2	22 (out of 9)

Out of 2 cases of Rho (D) isoimmunisation, one case was ABO blood group compatible pregnancy in AB group mother and the other was ABO blood group incompatible pregnancy in '0' group mother, who did not show evidence of isoimmunised mothers and results of direct antiglobulin tests are shown in table III and table IV.

Table III

Showing titres of immune anti A in 2 group 0 mothers and results of direct Coombs test with blood of newborn babies.

Case Srl No.	Titre of Immune anti–A	Direct Coombs test
20	4	negative
21	16	negative

Table IV

Showing titres of anti Rho (D) antibody by various methods in 2 maternal blood and results of direct Coombs test with blood of newborn babies.

Mother	Saline agglutination at 20 C	Albumin agglutination at 37 C	Enzyme agglutination using papain cystein at 37°C	Indirect antiglobulin method	Direct Coombs test
AB group	0	256	64	16	Positive
0 group	0	32	16	8	Positive

COMMENTS

In heterospecific pregnancies the isoimmunisation occur as a result of transplacental bleeding at about the time of delivery. Ascari et al (1969) concluded from their observations that maternal Rh isoimmunisation almost exclusively occurs after delivery. The foetal red cells can be detected in maternal blood which provide the antigenic stimulus for formation of antibodies in maternal circulation. The transfer of the maternal antibodies to the feetus through placental barrier may cause destruction of foetal red cells resulting in anaemia, jaundice and sometimes foetal deaths commonly grouped together as haemolytic disease of newborn.

In the present series only four cases showed evidence of isoimmunisation out of 20 cases of heterspecific pregnancies which is only 20% and appears to be much less in incidence when compared with 18% of cases of blood group incompatibility found by Bajpai et al (1971) in their series of 50 cases of neonatal jaundice as a result of known and unknown causes. Cases of neonatal jaundice due to other known causes diagnosed by other investigations, even associated with heterospecific pregnancies, were excluded from this series studied by the author. The findings of two cases of ABO iso immunisation in the present series of 11 ABO incompatible multiparous pregnancies indicate higher incidence than that found by Woodrow and Donhoe (1968), who recorded that ABO incompatible pregnancies were responsible for 1 in 35 instances of primary immunisation resulting from first pregnancies. The higher incidence in the present series may be due to multiparity of women.

Ascari et al (1969) reported that ABO incompatible pregnancy suppresses Rh immunisation and recorded 9% to 13% of Rh immunisation in O group mothers and 17% of Rh immunisation in non-O group mothers. The present findings of 1 case each in O group mothers (4 cases) and non-O group mothers (5 cases) does not differ much from the observations of above authors, except that in the present series no suppression of Rho (D) immunisation is appreciable, the reason may be less number of cases. Though it is well known that elution of anti A or anti B from the cells was the method of choice to detect sensitisation, but Haberman et al (1960), Yunis and Bridges (1964) demonstrated that elution is possible from those cases which give positive modified direct antiglobulin tests (Rosenfield, 1955), which was carried out in these cases. Inspite of the fact that in one case the titre of immune anti A was 16 in maternal blood by using indirect antiglobulin method which was as high as one of the cases of Rh sensitisation, the modified direct antiglobulin test in ABO incompatibility remained nagative (Tables 3 & 4).

SUMMARY

The reported series of 20 cases of neonatal jaundice associated with ABO and Rho (D) blood group incompatibility revealed evidences of iso-immunisation only in

4 cases. The red cells of newborn babies delivered by ABO immunised mothers did not show any evidence of sensitisation.

ACKNOWLEDGEMENT

I am thankful to Lt Col S J LUXA, Associate Professor and Officer-in-Charge Blood Transfusion Department, Armed Forces Medical College, Pune for guidence to publish this paper.

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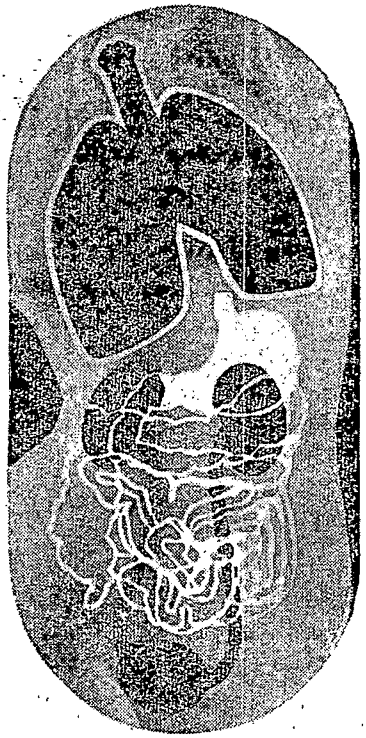

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THE RENAL PATHOLOGY OF HEATSTROKE

BY

Maj A. K. Pal* & Col S. K. Chopra**

INTRODUCTION

Malamud et al. in 1946 published their classical work on the autopsy material of 125 cases of heatstroke. Afterwards, since there has been no similar work either in India or abroad we undertook a study of the fatal cases of heatstroke. The clinical, haematological and biochemical features of these cases have been reported previously (Pal and Chopra, 1973). The present paper deals with our experience on the morphological changes in the kidneys.

MATERIAL AND METHOD

The material consisted of autopsy protocols (and in some cases also clinical protocols), paraffin blocks and stained and unstained sections of tissues from 41 fatal cases of heatstroke. The kidneys from 12 cases were available. Additional sections were prepared from the available blocks and the kidneys. All sections were haematoxylin and eosin stained. The morphological features of the kidneys were correlated with clinical and laboratory data as noted in the protocols.

OBSERVATIONS

The kidneys were overweight (combined weight of both kidneys-360-420g.) in 6 cases. Petechiae were noted on the outer surface of the kidneys in one case. Cut sections showed hyperaemia, more in the medulla in many cases. The microscopic changes apart from congestion seen mostly in the medulla in many cases, were degeneration of the proximal and distal tubules in 16 with blood casts therein in 4 cases (Fig. 1) and distal tubular necrosis in 7 cases (Fig. 2). The latter was seen in cases who survived 6 hours or longer. In 5 of these cases blood pressure readings at the time of admission were available; it was 80/60mm of Hg in 3 and unrecordable in 2 cases. In 4 patients the glomeruli were enlarged and slightly hypercellular in 2 of which some of the glomeruli were adherent to the Bowman's capsule (Fig. 3); the tubules were not

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involved. One of these cases showed focal mononuclear infiltration. In 3 patients marked medullary oedema and in one perirenal haemorrhage were observed but without any glomerular or tubular lesion. One of the patients with tubular degeneration showed fibrin thrombi in the interlobar vein (Fig. 4). In 11 cases there was no abnormality other than congestion

DISCUSSION

It is obvious from the present study that the kidney is damaged in majority of the cases of heatstroke. The same has been observed in the autopsy material by others also (Malamud et al, 1946, Ghore and Isaacson, 1949). The study of clinical and laboratory data (Austin and Berry, 1956, Kew et al, 1967, Shibolet et al 1967 (and renal biopsy (Kew et al, Loc. cit.) have afforded evidence of renal damage in nonfatal cases also. However, the changes are usually of mild to moderate degree and reversible. Of the 4 cases of Kew et al (loc. cit.) renal biopsy 6 months after heatstroke with acute renal failure showed normal kidney in 2, mild interstitial fibrosis in 1 and chronic pyelnephritis in 1 case. The latter was attributed to catheterisation. In none of our cases renal failure dominated the clinical picture and therefore could not be considered the sole cause of death though this has been reported by others (Austin and Berry, loc. cit.). It should be noted however, that kidney damage, though not necessarily severe, may contribute to death by augmenting hyperkalaemia which usually occurs during later stages due to widespread tissue necrosis, metabolic acidosis and destruction of sequestrated heat-damaged red cells (Baxter and Teschan, 1958).

An interesting finding was the presence of fibrin thrombi in the interlobar vein. Shibolet et al.loc. cit) noted this in one of their 6 fatal cases which also showed infarction. Four of our cases also showed fibrin thrombi in the liver. Recently haematological evidence of Disseminated Intravascular Coagulation (DIC) has been described in cases of heatstroke (Meikle and Grabill, 1967, Weber and Blakely, 1969). Fibrin thrombi noted in the present study may be an expression of DIC.

The mechanism of renal damage in heatstroke appears multifactorial. Hyperthermia can injure the kidney directly. Renal ischaemia due to hypotension plays an important part. Most of our cases suffered from hypotension from the initial stage. The ischaemic effect is aggravated by increased demand for oxygen due to hyperpyrexic hypermetabolic state. Products of other demaged tissue also put additional burden on the kidneys.

SUMMARY

The pathological changes in the kindneys in 41 fatal cases of heatstroke were studied and correlated with clinical and laboratory data. Evidence of renal damage was found in most of the cases. Degenerative changes in the tubules were seen in 16

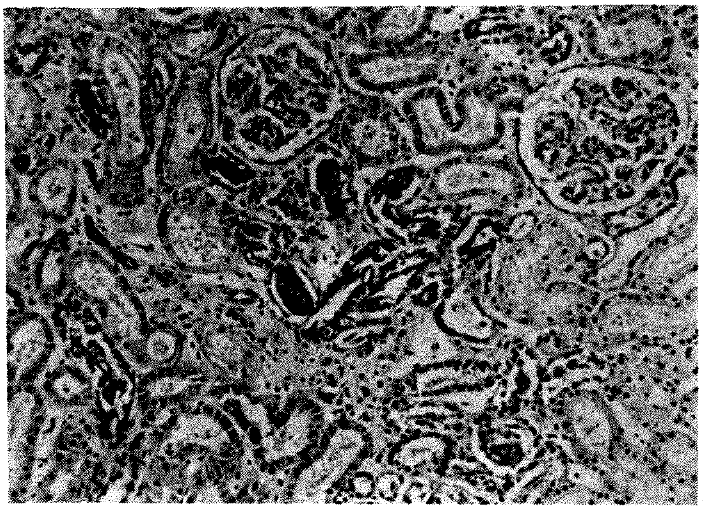

Fig. 1 Blood casts in the tubules. Period of survival–24 hours.
H. & E. Stain. X 120.

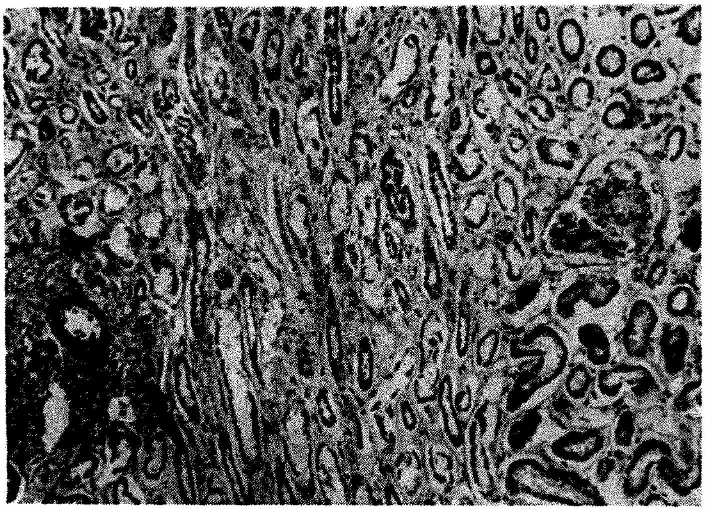

Fig. 2 Acute tubular necrosis. Period of survival–6 days.
H. & E. Stain. X 120.

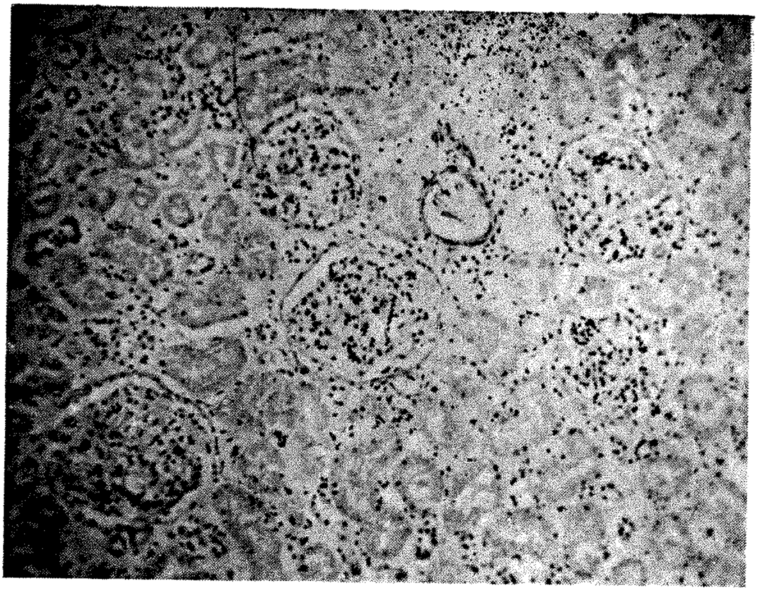

Fig. 3 Slightly enlarged and hypercellular glomeruli adherent to Bowman's capsule. Period of survival-4 days. H. & E. Stain. X 120

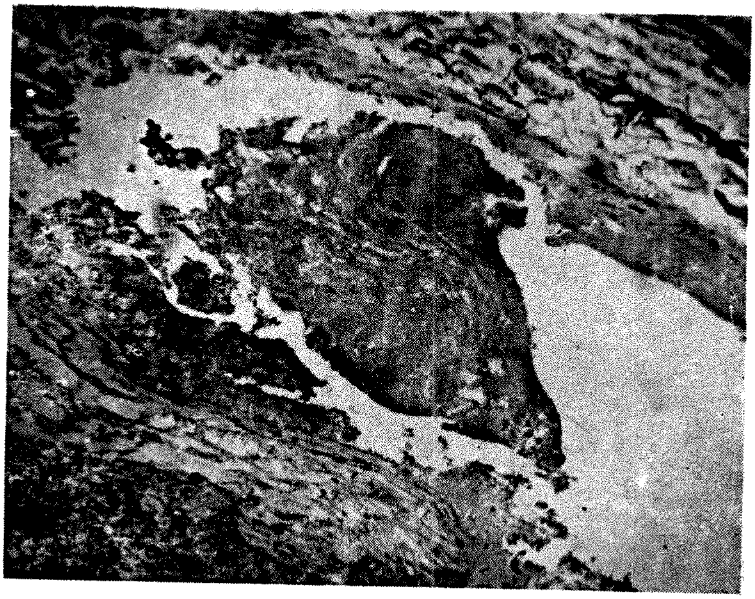

Fig. 4 Fibrin thrombus in interlobar vein. Period of survival ± 6 days. H. & E. Stain. X 120.

cases, distal tubular necrosis in 7 cases and glomerular involvement in 4 cases. One patient showed fibrin thrombi in the interlobar vein which are regarded as expression of DIC.

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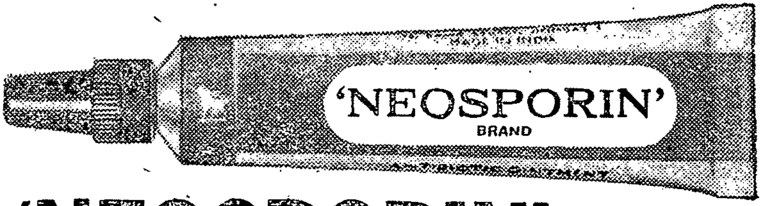

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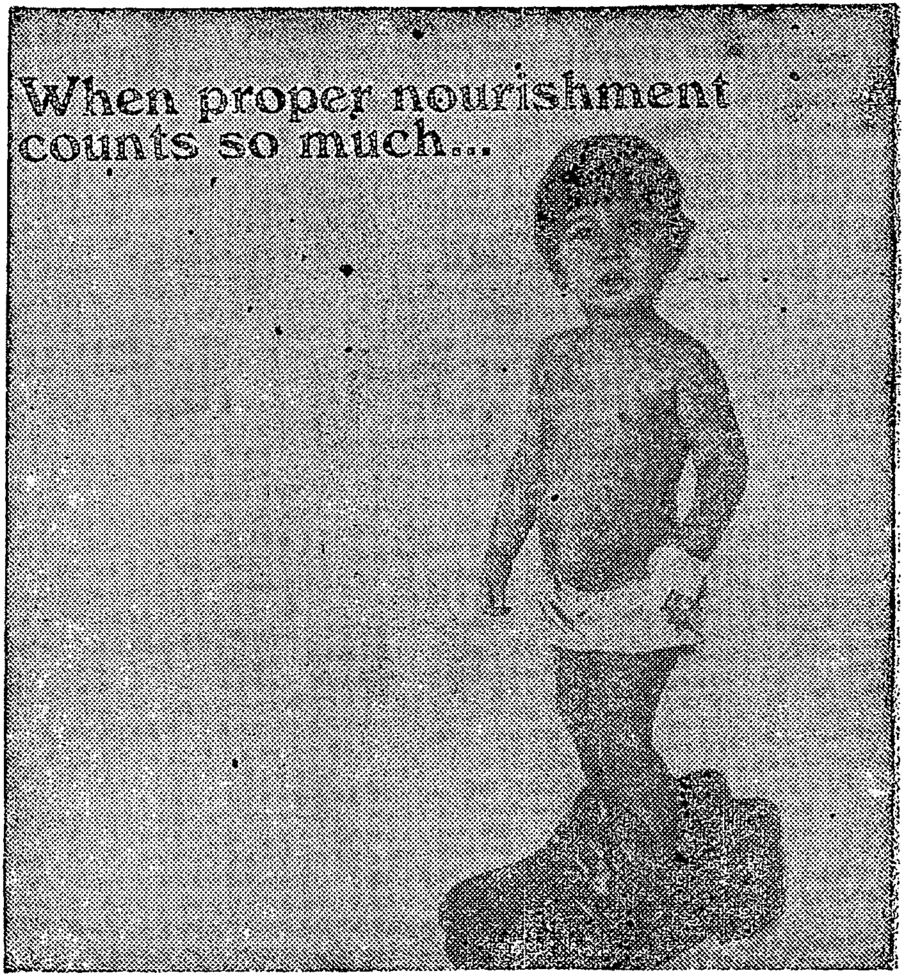
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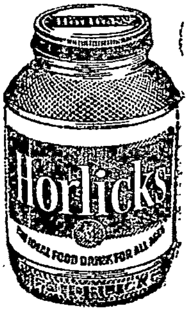

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A FORTNIGHT OF WAR SURGERY

BY

Capt (Miss) A Manorama, MNS*

It was 3rd December 1971, I was on long duty in Acute Surgical Ward of Military Hospital Ferozepore, as a reliever to sister-incharge. At about 1800 hours, there was a sudden black-out in the whole Cantonment Area. After few minutes, I heard a sound of heavy gun fire. Curious, I came out in the front verrandha, and found flashes behind the trees. A patient from Arty Regt told me that this firing was from the 'Hussainiwala Border' and seemed to be from the enemy side.

I thought for a while and then turned to my duty room. I rang up the duty ORA asking him to keep the operation theatre ready for any casualties. At about 2000 hrs, O.M.O. called me and told "Sister, come along with me, a casualty has come already in the MI Room." We both went to the MI Room found a 30 years old Havildar, having a big shell wound of the left forearm, bleeding profusely. His belongings and other valuables were deposited in the MI Room and fresh clothes were given to him.

I took the patient straight to the O.T. in an Ambulance. Within no time Surgeon, Anaesthetist and all ORAs reached the O.T. I.V. glucose 5% started after taking a sample of blood for grouping, cross matching, and Hb% estimation. We could not give him Inj. Morphia, as he came with full stomach. Inj T.T. ½cc I.M. stat was also given. I washed up to assist the case and kept the trolley ready. Toilet of the wound and debridement was done under local anaesthesia. The limb was supported on a crammer wire splint. Hourly T.P.R. and B.P. were recorded. I.V. fluids were continued and patient was put on antibiotics, and Inj Pethidine 100 mgm was given to relieve his pain.

Next case, which we received was a gun-shot wound chest left side. Patient was in agony and was dyspnoeic. He was restless and his pulse was very rapid. Inj Morphia 1/6 gr I.M. was given. Oxygen inhalation was started. Blood for grouping, cross matching and Hb estimation were taken and demand for blood was sent. I.V. 5% glucose drip was started. Surgeon explained to me that he was a case of Haemopneumothorax, and to keep a trolley ready for intercostal drainage under water-seal. Patient was put in position over an adjustable stool, which was improvised locally. Under local anaesthesia intercostal drainage with water seal was done at 8th intercostal space. After cleaning and applying acriflavine spirit, wound was dressed and sealed. The initial wound was repaired. Patient was kept in propped up position and good nursing care was given in O.T. as well as in the ward. Treatment on usual line was given, and patient became alright on 10th day.

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Third case was a shell wound left hypochondrium. Patient was in pain, cyanosed and in deep shock. He was severely dehydrated. Temp was 96°F, pulse 160/pm and BP 60/ Inj. Morphine ¼ gr I.M. was given, oxygen inhalation was started. I.V. glucose 5% started and was given in running drips. Blood sample for Hb%, grouping and cross matching was taken and demand was sent for blood. Catheterization was done and urine was sent for Lab investigations. Ryle's tube passed and gastric-aspiration was done. Patient was kept in pre-operative room for resuscitation, which was very important before anaesthesia and operation. All resuscitation measures were carried out. After 1½ hours or so B.P. came to 100/60. Patient was taken to operation table. I was ready with my trolley to assist. Exploratory Laparotomy was done under General Anaesthesia. Whole abdominal cavity was found full of blood. It was mopped quickly and carefully by the susgeon. Spleen was found ruptured. Five small perforations were detected in the small intestines, which were repaired in two layers. A big wound of descending colon with rupture of the outer coats, were found. A simple loop Colostomy was done. Abdomen was closed in layers, keeping a drainage tube. Patient was put on Inj Reverine I.V. and IM. antibiotics. Resuscitative and post operative treatment was carried out in the ward. The patient recovered after a month.

Another four cases of shell wound with minor injuries were waiting and were treated with debridement of the wounds and suturing etc. done accordig to the wounds.

The Matron sent a Nursing Officer to relieve me for breakfast as I missed my dinner previous night, then I realised that it was already morning, and 4th of December. I had a quick wash and after my break-fast when I returned at about 0800 hrs, O.T. was full of casualties, nearly 35 patients. Priority was given to shell wound chest, and maxillo-facial injury, while Gun-shot wound abdomen and missile wound with compound and comminuted fracture of femur were treated for shock.

All minor wounds were excised and sutured accordingly in the plaster room, by other surgeon and anaesthetist, who were posted from Military Hospital CHANDIGARH for this purpose. All required POP; and POP slabs were given. Out of major cases, two laparotomy, three interostal drainages, three amputations (one upper limb and two lower limbs) one burr hole, two debridement of the wound and skin traction in compound comminuted fracture of femur, one tracheostomy and four debridements of major wounds of chest, abdomen and thigh were done. Rest were all minor cases. Some how we managed to finish cases at about 1730 hrs and had our lunch in the O.T.

Our all clothing, dressings etc. were finished. Dhobi was called and all dirty linen were given to him and washed one's were received from him. Urgent indent was brought from the medical stores. That night we all were busy in replacing articles–packing operation towels, gown and gloves; making dressing, cutting and folding gauze pieces etc. and autoclaving them.

On 5th December 1971, half of the O.T. staff was sent to Faridkot to open surgical team's equipments and to function as separate OT at Faridkot, which was initially an old JCOs Mess.

On 5th December 1971 we had only 10–15 minor cases. After that we started changing dressings of all old patients which were to be transferred to Faridkot that evening. Patients with Thomas splint, were given Tobruk plaster. After sending the patients we packed up our O.T. in cold pitch dark night. I arranged 8 boxes, containing lotions, Bandages, Suture materials, POP dressings, surgical instruments and linen. Each box was given a number and duplicate lists were made for each. One was kept inside the box and another was filed in the inventory. With the help of ORA's; Diathermy, Suction apparatus, Boyle's apparatus, Electric sterilizer for instruments and other important items were packed very carefully. On 6th December at 0800 hrs, we loaded all our packages along with all other necessary articles, and our personal kit in the civil truck and started for new OT.

At about 1100 hrs, we reached Faridkot. In the new operation theatre, there were two rooms in the centre with well fitted shadow-less lamp and operation table in each. Right side, there were three small rooms as washing room, instruments room and autoclaving room. Outside there were two rooms, one was used as a store room and other was for eye-specialist. In front on either side there was a room, one for Surgeon and Anaesthetist and other as pre-medication room and plus sister's duty room, with 20 beds arranged in two rooms. After repeated carbolization we arranged all the articles in their places. In the evening we had 5-6 cases of different types of fracture, and were treated accordingly. At night, I went to my new room to have rest. I was awefully tired, as for the last 4 days and nights continuosly I was in the operation theatre. On seventh December, again we had a mass of casualties-about 38 in number. Most of them were having major shell wounds of back, thigh and lower extremity with fracture of the bones. Two of them were Gun-shot wound abdomen. Exploratory Laparotomy with Caecostomy was done on them. Three intercostal drainage and one orchiotectomy with perineal repair were done, one burr hole and two amputations below knee were done.

Though, we received patients of Gun-shot wound abdomen with a loop of intestine protruding out; head injury with grey matter of brain bulging out, but the most serious and horrible case, which I felt was the patient with Maxillofacial injury, struggling for breath, bleeding profusely, unable to talk and spitting out the blood due to fracture of maxilla with loose teeth. No Morphia was given to him due to respiratory obstruction. Immediately suction was started to prevent further swallowiog of blood into the trachea. On other hand, tracheostomy was done by the surgeon and tube was fixed and tied around the neck. After doing proper suction through the traceostomy tube, oxygen was started with a rubber catheter. Then mouth and throat were cleaned thoroughly with swabs and suction. A small pack was given for few minutes on the bleeding site and bleeding points were ligated. His loose dentures were placed in position and interdenture fixation was done by wiring. No bone fragment was removed. The oral mucus membrane was closed with fine chromic catgut. The wound of the cheek was repaired as usual.

An ORA was put on special duty to him. All care for tracheostomy tube was carried out. Oxygen inhalation was given intermittently. Mouth was attended periodically; lips and nostrils were lubricated to prevent fissures and sores. A small piece of jaconet was used to protect the dressing of the wound, owing to the dribbling of saliva. Two days I.V. fluids and two days intra-nasal gastric feed was given. Afterwards he was put on 3000 calories diet orally. 2 hourly milk, fruit juice, thin custard, egg flip, soup and glucose water given. He was taught to clean his mouth after each feeding. On 4th day Tracheostomy tube was removed. Pin sutures were removed after 7 days. Antibiotic was continued for 10 days.

On 8th and 9th December, we received about 12-14 cases per day. Most of them were shell wound and Gun-shot wound of lower and upper extremities and two to three foreign body in eye and ear.

The most important days were 10th and 11th December, when we worked continuously without a break. There was no time at all to have our food even. We received different types of cases, like head injury, mine blast injury of hand, Gun shot wound (GSW) of abdomen, leg & chest, and patients with multiple injuries. During these two days, we received about 58 cases. Out of that 30 cases were operated.

We had one interesting case. Sepoy Dhani Ram of 5 Dogra Regt came with Gun-shot wound in the abdomen. The enterence wound was in the abdomen and the wound of exit was in the thigh. He was in severe shock and general condition was also poor. He was a thin lean young boy. After resuscitation, when abdomen was opened, seven perforations were found in the small intestine, one in the bladder and slight injury to the rectum and after that the tract was leading to the posterior aspect of the thigh. After repairing all perforations, abdomen was closed in layers, keeping a drainage tube. The wound in the thigh was attended as usual and dressed, urinary catheter was passed and continuous drainage done for healing of the bladder. Blood and other I.V. fluids were given for 8 days. Inj Reverine & I.M. antibioties were given along with other supportive treatment. All post-operative care was carried out in the ward. Oral feed started on 8th day with weak tea and fruit juices. Sutures were removed on 11th day. Dressing was done regularly. Secondary suturing of the thigh wound was done after 40 days. After removing sutures on 9th day, patient was sent on sick leave.

Another interesting case was a Gun-shot wound chest Rt. The bullet had entered from 2nd intercostal space and after piercing 3rd 4th 5th, 6th 7th ribs, lung and part of diaphragm, came out from the 8th intercostal space. Inter costal drainage under water seal and strapping of the chest was done. The external wound was repaired. With all resuscitative measures, I.V. fluids heavy antibiotics and with good nursing care, patient improved rapidly.

Afterwards we used to get only mine blast injuries, and some, vehicle accidents and other war accident cases. But still we were quite busy in looking after our post operative cases. During this period in managing our cases, we have been facing so many difficulties. As there was not proper post-operative room, and only tent was fixed, we had to undergo many difficulties to save patients from chill and infection. The area around the O.T. was full of dust. We covered it with hey.. With short staff of ORA we had to look after the dressing of all wards, as none of the wards were having proper dressing room. There was short supply of water, and electricity off and on. We were working with emergency light and we were preserving water for emergency use. Upto 20th December, night time, I was sleeping in the OT itself in my duty room, so that immediately I can help in attending the emergency cases, without delay in calling me from my room. We had small bath room close to our OT for Officers. There I used to finish my toilet and changing early morning. Most of the time I was in my OT Dress, otherwise we were wearing O.G. dress with peakcap. There was no long and short duty for one month. One nursing officer was given in OT to relieve me for meals and to help in other work. Mess was just behind the OT so whenever I got time I used to run and have my meal. I often tried to go to my room to have bath in the afternoon, as no warm water was available we had to have cold bath only. After 17th of December cold surgery started as usual and I started working in acute surgical wards in the evening. On 18th December all casualties were calculated, as undermentioned list :-

Shell wound	–	104
Mine Blast	–	19
Grenade	–	10

Gun shot	–	70
Bayonet	–	6
Misc	–	11
Total Cases	–	220
Sitting	–	103
Lying	–	117

Total Operations : 183

	Major	Minor
Upper Limb	34	3
Lower Limb	58	18
Abdomen	10	–
Chest	14	–
Maxillo-facial	5	11
Skull & Scalp	3	2
Eye & Ear	3	–
Multiple-injuries	22	5

We came back to FEROZEPORE on 9th Mar 1972.

ROLE OF O. T. TEAM DURING INDO-PAK CONFLICT 1971 IN MILITARY HOSPITAL JULLUNDUR CANTT

BY

Major (Miss) G. Sylva, MNS*

INTRODUCTION

The great litrary genius Oscar Wild has said that "Experience is only a series of mistakes done and having realised it". Lessons learnt by successive wars after Independence, namely Goa operations, 1962 war with China, Kutch operations and Indo Pak conflict of 1965 had put us on a better footing to deal with war casualties of 1971.

The role of operation theatre team in management of war casualties based on the author's experiences during Indo-Pak War 1971 at Military Hospital Jullundur Cantt is presented in this paper.

The subject has been discussed under the following heads:—

(a) Planning

(b) Training of personnel

(c) Equipment

(d) Management of casualties

(e) Conclusion.

Planning

Planning and preparation started as early as Aug 1971. The usual doctrines of planning were resorted to, namely :—

(a) flexibility of plan
(b) adequate reserve
(c) mobility of resources

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The planning was aimed at receiving 200 casualties per day. Bed occupancy of war casualties were thought of and elective surgery which could be postponed without undue discomfort to the patient were all cancelled.

Training

Training of personnel was most important. Many did not have experience of war surgery. Regular lectures for theatre staff were arranged which included ORAs, student nurses, and nursing officers. Many ORAs who were trained in this centre were posted out to Field areas where they could keep up with the war casualties because of this training. They developed confidence in dealing with any type of war injuries and could be of great help to the surgeons.

The training was war oriented. The doctrine – "The more we sweat in peace less we bleed in war" was followed and implemented. The student nurses and General Duty Nursing Officers were trained to look after injured round the clock. Stress was laid on :—

(a) adaptability

(b) resourcefullness

(c) enthusiasm

(d) stamina.

Acute shortage of trained theatre sisters was anticipated and to overcome this drawback, four newly commissioned Nursing Officers were given ad-hoc trainig in theatre duties. This forethought paid handsome dividends when casualties actually started pouring in.

Blackout practices were carried out as a routine and theatre personnel were well trained to carry out their duties during enemy bombardment. Blackout of theatre and protection against splinter wounds to patients by sand bags were practiced.

Equipment

Equipment of operation theatre is the responsibility of theatre matron. The requirements to meet with war - casualties were well anticipated. Theatre linen was a problem. Other materials included suture materials, splints, plaster of Paris, infussion fluids which included Ringer's Lactate solution and Haemaccel. Blood was no problem with enthusiastic patriotic local population. Steriliser drums were in acute shortage and it was overcome by wrapping operating gowns and towels in bed sheets and

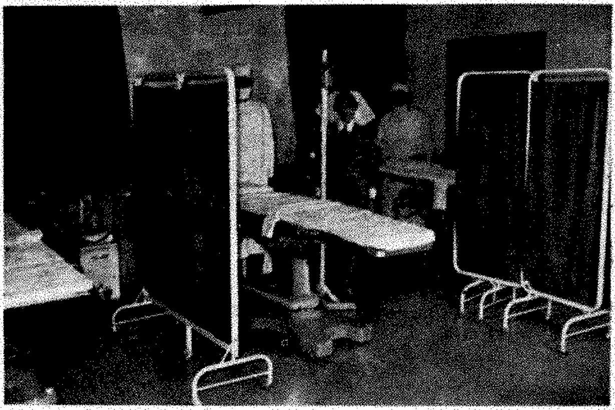

Fig. 1 A view of the improvised operation theatre.

surgical dressings in double pillow slips and directly autoclaving them. Anaesthetic equipment were adequate and posed no problem.

Management of Casualties

Triage was followed even after reception in the operation theatre. At a time upto 30 cases were waiting for life and limb saving surgery. In the pre-operative room transistors were provided to the patients who could hear to nice luring music or to the news about their comrades fighting valiantly on the battle front, which boosted their morale considerably.

On 5 Dec 71 casualties started pouring in. Many needed active resuscitation and good surgical toilet. The plaster room was converted to full fledged operating room and three tables, each with a Boyles apparatus and a suction apparatus were provided. (Fig 1). Main theatre were reserved for major thoraco-abdominal and neuro-surgical cases. This arrangement excluded any possibility of cross infection and it was gratifying that there was not a single case of tetanus or gas gangrene.

By the time the casualties started coming in, the hospital was left only with skeleton staff or ORAs.

During this period the surgical and anaesthetic workloads were as follows :—

Surgical work-load	
Major operations	67
Surgical toilet	546
F.B. removal	54
POP applications	296
Anaesthetic work-load	
General anaesthesia	537
Spinal	14
Local	599

This heavy load of work was carried out only because of determination, enthusiasm and initiation. It is of interest to note that one of the officers wife who is a nurse trained in UK volunteered and only with one ORA to assist her, managed the instrument room throughout maintaining a regular supply of instrument trolleys and working round the clock.

CONCLUSION

The following suggestions are offered for the future :—

(a) Preparation during peace is essential for smooth and faster turn of work during war.

(b) Co-ordination between surgeon, anaesthetist, theatre matron and ORAs for effective and smooth function of O.T.

(c) Available personnel including civilians who have some background training in medical work can be utilised provided they are actually incorporated in the operation theatre team well in advance and properly briefed.

(d) While the patients are waiting their turn in the operating room to be attended to, personnel and human touch from members of the O.T. team as well as soft music to break the monotomy may go a long way to keep the morals of the wounded jawans high.

CARCINOMA OF THE EXTRAHEPATIC BILE DUCTS

BY

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INTRODUCTION

Neoplasm of the extrahepatic bile ducts is rare and produces more often than not problematic cases from both diagnostic and therapeutic angles. The present paper deals with 3 such recently encountered cases and one from our old record.

CASE REPORTS

Case 1

A male aged 27 years was admitted on 30th April 1966 with complaints of pain in the right hypochondrium for 7 months, a lump in the same region for 4 months and progressive jaundice for 2 months. The patient was emaciated and deeply jaundiced. The liver was enlarged 6 cm downwards, irregular, firm and tender. The gall bladder was also enlarged and tender. There was ascites. Investigation – Serum bilirubin – 20 mg per 100 ml. Liver biopsy showed features of bile ducts obstruction. At laparotomy, performed on 18th hospital day, the liver and gall bladder were found enlarged. All the structures in the porta hepatis and in lesser omentum were adherent to one another. Cholecystojejunostomy was performed. A wedge biopsy of the liver showed again features of bile ducts obstruction but no tumour. The patient's condition deteriorated after a temporary phase of improvement and he expired on the 40th hospital day. The clinical diagnosis was obstructive jaundice.

Case 2

A female aged 60 years was admitted on 31st Jan 73 for jaundice of one month duration. The patient was slightly emaciated and deeply jaundiced. The liver was enlarged 4 cm downwards, slightly irregular, firm and nontender. There was mild ascites. Investigations :- Hb- 9 G%. TLC and DLC - normal. Serum bilirubin - 16.5 mg per 100 ml, conjugated bilirubin-10.9 mg per 100 ml. Serum proteins-normal. Thymol turbidity 7 U. Thymol flocculation - positive. Serum alkaline phosphatse - 80 to 280 KAU per 100 ml. SGOT - 341 IU and SGPT - 401 to 1180 IU per litre. X-ray of the chest and abdomen and barium meal were normal.

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The clinical diagnosis was carcinoma of the liver. She was treated conservatively to build her up for laparotomy but died on the 29th hospital day.

Case 3

On 14th May 1973 a 60 year old female was admitted with jaundice and right hypochondriac pain for 4 months. She was anaemic and deeply jaundiced. The left supraclavicular lymph nodes were enlarged. The liver was enlarged 7 cm downwards, firm, smooth and tender. The gall bladder was also enlarged. Investigations :- Hb-9 g%. TLC - 21800/cmm, neutrophils - 96%. Serum bilirubin 13 mg to 24.6 mg per 100 ml with conjugated fraction 6.4 to 16.3 mg per 100 ml. Thymol turbidity - 12 U. Thymol flocculation - positive. Serum alkaline phosphatase - 150 to 180 KAU per 100 ml. SGOT - 39.6 and SGPT - 54.2 IU per litre.

The clinical diagnosis was carcinoma of the liver and/or cholelithiasis.

At exploratory laparotomy, performed on 13th hospital day, one gall stone was removed from the gall bladder. No growth could be detected anywhere. The patient died on the 4th postoperative day.

Case 4

A female aged 30 years entered the hospital on the 7th Jan 73 because of generalised abdominal pain, pruritus and occasional fever of 6 weeks duration. She developed jaundice 3 years ago which lasted only 15 days. The patient was very pale. The liver was enlarged 5 cm downwards, smooth and tender. After 10 days she developed jaundice and fever of 101°F. The fever (101 to 104°F) continued until death. After about 5 months the gall bladder became palpable and it was tender. The liver by then enlarged 1 cm more. There was haematemesis once and melaena twice during the whole illness. Investigations :- Hb - 9 g%. Serum bilirubin - 0.6 to 11.8 mg% with conjugated fraction 1.1 to 7.8 mg per 100 ml. It became normal 3 times. Serum proteins - normal. Thymol turbidity - 3 to 10 U. Thymol flocculation - positive. Serum alkaline phosphatase - 4.2 to 83 KAU per 100 ml. SGOT - 15 to 95 IU and SGPT - 21 to 240 IU per litre. X-ray of the chest and abdomen and barium meal were normal. Liver biopsy showed cholangiolitis and hepatocellular cholestasis.

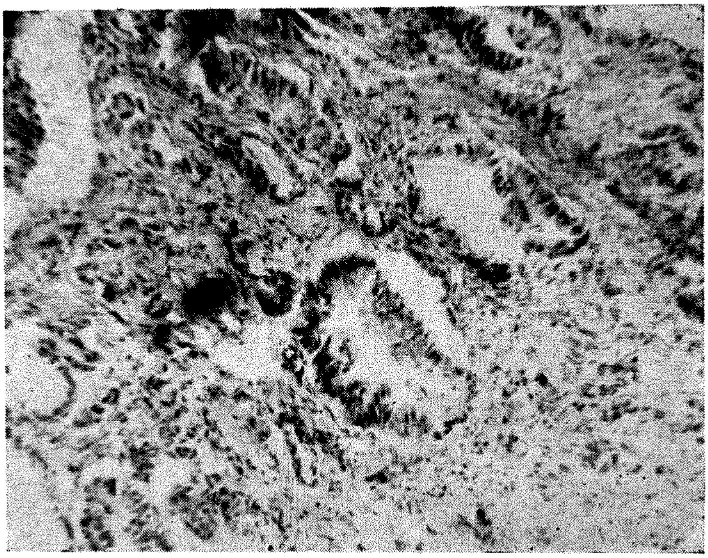

Fig. 1 Scirrhous carcinoma of the common hepatic duct.
H. & E. Stain, X 180

The clinical diagnosis was chronic active hepatitis, cirrhosis of the liver or carcinoma of the liver. She wes treated with several blood transfusions, corticosteroids and many broad spectrum antibiotics. She died on the 180th hospital day.

AUTOPSY FINDINGS

Case 1

Small nodular growth were seen in the liver. The whole of the extrahepatic biliary tract including the gall bladder was involved in a diffusely infiltrating neoplastic process. Microscopically, sections from different parts of the biliary tract showed scirrhous mucin secreting adenocarcinoma. Metastases were noted in the lymph nodes in the porta hepatis, in the liver, suprarenals and the right lung.

Case 2

There was a spindle shaped swelling of the common hepatic duct and thickening of the wall of the adjacent part of the common bile duct. The liver showed a small greyishwhite area adjacent to porta hepatis. Microscopically the growth was scirrhous adenocarcinoma (Fig. 1), as in case 1. The greyishwhite area in the liver showed metastases.

Case 3

The common bile duct and the cystic duct were irregularly thickened and the gall bladder showed two small raised areas. The liver showed a few small nodules. Microscopically the common bile duct, cystic duct and gall bladder showed poorly differentiated adenocarcinoma. Also the terminal part of the common bile duct, pancreatic duct, ampulla, second part of duodenum and head of the pancreas showed the same type of adenocarcinoma. Metastases were noted in the liver, kidneys, lungs and mesenteric and supraclavicular lymph nodes.

Case 4

The terminal part of the common bile duct, pancreatic duct and ampulla were blocked by friable growths. The whole of the extrahepatic part of the biliary tract was dilated. The liver showed a right lobel abscess (6x5 cm). Small polypoid projections were seen around the papilla. Microscopically there was adenocarcinoma involving the terminal part of the biliary tract and second part of the duodenum. The liver abscess was a cholangiolitic abscess.

DISCUSSION

The incidence of carcinoma of the extrahepatic bile ducts, contrary to general impression, equals that of carcinoma of the gall bladder and pancreas (Kirshbaum and Kozoll, 1941, Willis, 1960). In a series of 13300 autopsies in the Cook County Hospital, Chicago Kirshbaum and Kozoll (loc. Cit), found carcinoma of the extrahepatic bile ducts in 62, gall bladder in 55 and head of the pancreas in 41 cases. The sites involved in the former were common bile duct in 32, hepatic ducts in 13, papilla in 10 and cystic duct in 7 cases. Subsequently 26 more cases had been reported from the same hospital (Lee Stroh, Reed, Diffenbaugh and Anderson, 1963). It accounts for about 3% of all cancer deaths in the USA (Ham and Mackenizie, 1964). In a review of 570 cases collected from the world literature Sako, Seitzinger and Garside (1957) found the autopsy incidence to be 2.88 to 4.65% of all cancers. In our autopsy series of 1006 cases including the present 4 cases there were 2 cases of carcinoma of the head of the pancreas and none of the carcinoma of the gall bladder. The biopsy during the same period provided 9 cases of carcinoma of the gall bladder and 2 more cases of carcinoma of the head of the panereas.

The etiology of the disease is still unknown. There is associated gall stone in 29 to 55% of cases (Kirshbaum and Kozoll, loc. cit., Scott, 1966, Evans, 1968). - Bile duct cancer has been produced in experimental animals by feeding Thioacetamide (Gupta, 1955) and Aramite, an alkylating agent (Sternberg, Popper, Oser and Oser 1960) and by injection of carcinogenic extracts from bile and gall bladder (Steiner, 1942) and bile from patients with bile duct cancer (Fortner, 1955). Administration of Thorotrast (thorium dioxide) in man has been reported to be followed by the development of biliary tract carcinoma 11 years (Okinaka, Nakao, Ibayaski, Nakaisumi, Kakehi and Sugimura, 1957) and 17 years (Roberts and Carlson, 1956) after its administration. Development from papilloma of the bile ducts has been suggested by some (Dick, 1939 Boother and Pack, 1952) but refuted by others (Schmug, 1958). The contribution of papilloma, if any, is likely to be small since only 30 cases of papilloma have been reported (Ham and Mackenzie, loc. cit.).

The disease affects individuals mostly in the 6th and 7th decades of life, the youngest reported was 17 years old (Ham and Mackienzie, loc. cit). Our 2 patients were quite young 27 and 30 years of age respectively. Three out of 4 of our cases were female though a male preponderance is usual (Willis, loc. cit.). The patients usually present with progressively increasing jaundice with or without abdominal pain. Rapid onset of symptoms characterises the disease. Pruritus is a prominent feature which was present in only one of our cases. Pruritus preceding the onset of jaundice, as in case 4, strongly suggests extrahepatic obstruction. (Flandreau, Weber and Baird, 1957). Usually the liver is enlarged and the gall bladder is palpable in about 75% of cases (Bailey and Love, 1968). In our case 2 the gall bladder was not enlarged as the obstruction was in the common hapatic duct.

Correct clinical diagnosis is seldom made. Even operative diagnosis is difficult as illustrated by our cases. In the series of Kirshbaum and Kozoll (loc. cit.) only

22% of cases could be correctly diagnosed. Laboratory investigations help in establishing obstructive nature of the jaundice. Both serum bilirubin and alkaline phosphatase progressively rise. Fifty percent or more of the serum bilirubin prove to be of conjugated type. Intermittent obstruction with fluctuating jaundice may occur, as in case 4, in case of "Periampullary" carcinoma due to relief of obstruction by ulceration. Haematemesis and melaena, as in case 4, point to "Periampullary" carcinoma. For localisation of the growth the following investigations are helpful. Subnormal pancreatic function after secretion stimulation indicates involvement of both the common bile duct and pancreatic duct while normal values point to obstruction of the bile ducts (Beeson and McDermot, 1971). The cytologic study of duodenal aspirate has proved useful (Raskin, Wenger, Sklar, Pletcika and Yarema, 1958). Hypotonic duodenography may show duodenal deformity indicating neoplasia of the periampullary region. Percutaneous transhepatic cholangiography (Storer, 1966) is of great value. Oral and intravenous cholangiogram are unreliable in presence of marked jaundice. Operative cholangiogram (Kantor, Evans and Glenn, 1955, Evans and Mujahed, 1959) is indicated in certain cases where no lesion can be seen at laparotomy.

Results of treatment are very di couraging. In about 15% of cases the growth would be resectable (Bailey and Love, 1968). The average survival period after operation has been reported to be 4.2 months (Sako et al, loc. cit.). The tumour at the distal part of the biliary tract may lend itself to radical surgery like pancreaticoduodenectomy. Cholecystoenterostomy may prolong the life in inoperable cases.

SUMMARY

Four cases of carcinoma of the extrahepatic bile ducts have been reported. Three were female. The youngest patient was 27 years old. Jaundice was present in all cases and was fluctuating in one case. The latter had haematemesis and malaena. Pruritus was present in only one. The liver was considerably enlarged in all cases and gall bladder in 3 cases. The serum bilirubin, alkaline phosphatase and transaminases particularly, SGPT were high in all cases. The neoplasia involved the common bile duct, cystic duct and gall bladder in one, commom hepatic duct and common bile duct in one, common bile duct, cystic duct, gall bladder and periampullary region in one and only the periampullary region in one. Metastasis in the liver was present in 3 cases and distant metastases in 2 cases.

The etiology, clinical features, diagnosis and treatment of the disease have been briefly discussed.

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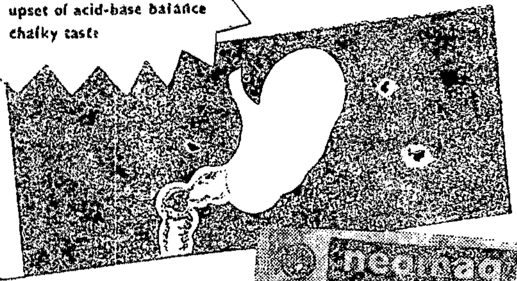

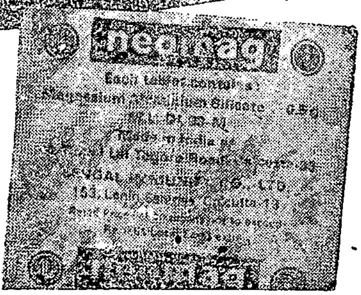

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INJURIES OF SPLEEN

(Case Reports)

BY

Lt Col S.K. Gupta*

INTRODUCTION

Injuries to spleen may result from a penetrating wound, as from a missile, direct external violence or a severe crushing. The smaller size of spleen renders it less vulnerable to missiles, but its friability and greater vascularity make it more susceptible to blows, crushes and blast. It is damaged in only about ten per cent of abdominal injury (A Field Surgery Pocket Book, 1962). Rupture of spleen has also been reported follwing a sudden increase in intra-abdominal pressure or unexpected and unguarded movements of the trunk. In such cases and in others ruptured by trivial injury, the spleen is frequently abnormal. Spontaneous rupture of the spleen has been described, but in most of the reported cases it has been established either that a history of injury has been for some reason suppressed or forgotten by the patient.

Bleeding from a spleen injured by a missile may cease spontaneously but is very apt to restart. Splenectomy is, therefore advisable in every case even though the damage seems trivial. Direct external violence may fissure the surface of the spleen or cause a deep split which may involve the hilum and be followed by profuse intra-peritoneal bleeding and profound shock. More often, the symptoms are biphasic. The injury which may be sufficiently severe to fracture the left tenth or eleventh rib. either contuses the convex surface of the spleen with the formation of a subcapsular haematoma or compresses the spleen against the vertebral column and causes a split along its posterior border. Slow bleeding occurs into either the subphrenic space or the retroperitoneal space between the layers of the lieno-renal ligament. Physical inactivity and the hypotension consequent upon the injury allow a clot to form which plugs the rupture temporarily. After an interval which may vary from a few hours to several months, some physical stress causes rupture of the subcapsular haematoma or loosening of the partially organised clot plugging the rent and free bleeding occurs in the peritoneal cavity. This pathological sequence explains the diverse ways in which injuries of the spleen may present and which has given rise to such descriptions as immediate, Biphasic, delayed and spontaneous rupture (Macpherson, 1969).

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*Specialist in Surgery, Military Hospital, Mathura.

CASE REPORTS

Case No. 1

Sep Baljit Singh of 23 BSF had sustained shell wounds on his back on 4.12.71 and when he was abmitted to MH AMRITSAR, signs of intraperitoneal haemorrhage were obvious but no wounds of exit. X-Ray of abdomen showed a large metallic FB in left hypochondrium. The urine obtained by cathetrisation and Ryle's tube aspirate were clear. Laparotomy revealed a large collection of blood in the peritoneal cavity, impacted metallic FB about 6 cm x 4 cm on the convex surface of the spleen, a tear in the adjacent part of diaphragm and perforations in the small gut. Splenectomy with repair of tear in the diaphragm and multiple intestinal perforations along with wound debridement of multiple shell wounds on the back were done. He had a slightly stormy postoperative convalescence with development of pelvic cellulitis due to a retained FB in pelvis, not visualised in the earlier skiagrams. However with conservative treatment pelvic cellulitis resolved and the laparotomy wound healed primarily.

Case No. 2

Ex NK Bhagwat Saroop aged 65 years was admitted to Military Hospital, Mathura on 29.8.72 with history of being knocked down by a steam locomotive, in a state of severe shock and profound respiratory distress. He had fracture of left 4,5,6,7, 8,9, ribs with double fracture of 4,5,6, ribs resulting in a flail chest and paradoxical respiration. No intra-abdominl mischeif was evident on clinical or radiological examination on admission. Closed external fixation by pressure with a firm pad corrected the paradoxical respiration and restored effectual pulmonary ventilation. Repeated examination did not reveal any intra-abdominal mischief and shock was overcome with IV fluids. However, persistance of tachycardia in spite of a well sustained BP and progressively lowering of Hb% and RBC count in association with the severe crushing injury of left side of chest without any evidence of intrapleural bleeding raised the suspicion of rupture of spleen. Laparotomy revealed a large collection of fluid blood in the peritoneal cavity with a large split along the posterior border of the spleen. Splenectomy was successfully carried out.

Case No. 3

Sep GS GIRI of Gorkha Regt was admitted to Military Hospital AMRITSAR on 22.11.71 with a history of accidental injury to left side of chest about 4 hrs. previously by a bullet from his own sten gun. When first examined after being brought to the hospital, his general condition was extremely good without any tachycardia, hypotension or any evidence of shock. The wound of entry was at left 6th interspace about 8 cm from midline and wound of exit at the post axillary line in the same space. There was no clinical or radiological evidence of any haemo or pneumothorax. There was, however, some muscle guard in left upper abdomen without any evidence of free fluid in the peritoneal cavity and bowel sounds were normally heard. Ballance's sign was not present as also Saegesser's sign (Aird, 1958). Plain X-Ray Abdomen did not reveal any gas shadow under the diaphragm, or any raising of the left dome and normal psoas shadow was seen. Blood examination revealed Hb% 16.0 gm with normal TLC and DLC count. In view of the persistance of the muscle guard in left upper abdomen observed on repeated examination of the patient during the next 2-3 hours, he was treated on conservative lines keeping in view the possibility of injury to spleen in spite of the absence of any clinical signs of intraabdominal mischief. After 48 hours of admission, there was no change in the general condition of the patient and the muscle guard in the left upper abdomen completely disappeared. Patient started insisting on solid food as he was feeling increasingly hungry. However, Hb% repeated after 48 hours was 12.0% and next day 10.0%. Repeated plain X-Ray abdomen did not reveal any abnormality except medial displacement of normal gas shadow of stomach under left dome of diaphragm. Exploratory laparatomy on 26.11.71 revealed a large blood clot in left hypochondrium surrounding the spleen which had a large laceration at the upper pole with a small tear in left dome of diaphragm, but no fluid blood in the general peritoneal cavity. Splenectomy and repair of the tear in the diaphragm was done in the usual way and no undue friability of the splenic pedicle was noticed. Patient made an uneventful recovery with primary wound union.

DISCUSSION

Three cases of injury to spleen which were successfully operated by the author are reported. The first case was a typical example of multiple intra-abdominal injuries by missile, causing penetrating abdominal wound. Laparotomy was obligatory in this case of penetrating abdominal injury and splenectomy was done as injury of spleen was detected. The second resulted from severe crushing injury and diagnosis was aided by the presence of fracture of lower ribs on the left side and persistance of shock in spite of closed external fixation and progressive lowering of Hb% and RBC count in the absence of any intrapleural bleeding. The third was a delayed type of case and the aim in such cases i.e. to diagnose the condition before the haematoma bursts was achieved by keeping the patient under constant observation with repeated blood cell and haemoglobin estimation and skiagrams of abdomen (Bailey, 1967).

The abdomen was opened by left paramedian incision in all the three and no difficulty was experienced in performing splenectomy by this route. The splenic pedicle was secured by a number of haemostats avoiding injury to tail of the pancreas, and each moiety of tissue was transfixed with a needle and ligature thus avoiding cutting out or breaking of a ligature. In all cases, a drainage tube was brought out on the left flank to minimise complication due to associated damage to pancreas. The laparotomy wounds healed primarily.

SUMMARY

Three cases of injury to spleen are reported and the diagnostic difficulties of each case discussed. All the three cases were successfully operated.

ACKNOWLEDGEMENT

I am extremely grateful to Major J.D.B. Bryant for anaesthetic management of first and third cases and to Major J.D. Pal for the second case.

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AN UNUSUAL CASE OF CONGENITAL TUBERCULOSIS

(Case Report)

BY

Capt (Mrs) B.K. Sohi* and Lt Col A.S. Sohi**

INTRODUCTION

Congenital transmission of tuberculous infection is rare and still rarer are the cases of congenital tuberculosis whose mothers are infection free. Only five cases have been reported in literature so far. Out of these five, mothers of four developed tuberculosis at later date, and all the infants died, (Hughesdon 1946). The case being reported now, is still alive and healthy at the age of one and a half year, and the mother is infection free upto date.

CASE REPORT

Sanjay a first issue was born at full term on 3rd Dec 1971. Mother noted a swelling in left axilla when the child was just fifteen days old (Fig. 1). On 3rd Jun 72, child was brought to the Family Health Centre, AMC Centre and School, Lucknow for treatment. On examination baby looked active with normal growth for his age. There was a softish circular mass size of a walnut near the anterior fold of left axilla. There was a BCG scar on the left shoulder, which was given when the child was four days old, without prior Mantoux testing. Lungs showed no abnormal finding clinically. Heart was enlarged clinically, apex beat being palpable in the sixth space at anterior axillary line. Percussion was not reliable due to thin chest wall of the infant. There were no murmurs, but heart sounds were distant even though the apex beat was palpable. Infant weighed 6.36 Kg. Temperature was normal. (Fig. 2).

Lab findings-urine; albumin trace, pus cells 3-5/hpf. Haemoglobin 11.5 gm%, TLC, 18000/cmm, polymorphs 30%, lymphocytes 70%.

Antitubercular treatment, (isonex+inj streptomycin combination) was started on 19th Jun 72, along with the antianaemic drugs. Lymph gland biopsy was done on

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5th Jul 72 and the mass in left axilla, which actually was a lymph gland, removed. Biopsy report came as 'tubercular lymphadenitis. (Fig. 3). By 20th Jul 72, heart size reduced considerably clinically, apex beat was localized in fifth space 1.5 cms outside the midclavicular line. He had gained one kg in weight. A draining sinus formed at the biopsy site and it healed by 11th Sep 72. (Fig. 4). By 18 Aug 72 heart size had come down to normal clinically, and heart sounds of normal intensity were heard. His milestones were normal.

From 26 Mar 73 injection streptomycin were discontinued and child was put on isonex+PAS combination, after nine and a half months treatment with isonex+streptomycin. Weight at this time was 8.1 kg. On 10 May 73 he weighed 10.9 kg. Clinically there was no abnormal finding in any system except enlarged post cervical glands and inguinal glands size ½ × 1 cms both sides. These were not palpable before. Antitubercular treatment is still continued and it is intended to complete one years treatment. (Fig. 5).

Latest lab findings – Erythrocytic sedimentation rate 16 mm/hr haemoglobin 10 gms%. TLC 11,000/cmm. Polymorphs 36% lymyhocytes 64%. X-Ray chest normal (Fig. 6).

Both the parents were investigated and did not show any evidence of tubercular infection. There was no history of tuberculosis in the family and neighbourhood. Mother's, ESR was 12 mm/hr, X-Ray chest normal and Mantoux test was negative.

DISCUSSION

Uncommonnes of congenital tuberculosis is evident from Debre's (1948) study, where out of 1369 infants born of tuberculous mothers none showed evidence of congenital infection over a period of four years. That was prior to the availability of wide range of antitubercular drugs. With the advent and availability of these drugs chances of having congenital tuberculosis are remote.

Cases of congenital tuberculosis whose mothers showed no evidence of tuberculous infection at the time of delivery are reported by, Straus, (1895), Morley (1929), Soderstrum (1932), Pagel & Hall (1946), and Hughesdon (1946). Except Hughesdon's case mothers of all other children developed tuberculosis later on. Usually symptoms start during the first month of life. These are anorexia, vomiting, loss of weight, raised temperature, sometimes enlarged spleen and liver. Cough and physical signs of bronchitis and pulmonary consolidation in some and extensive lung involvement with few clinical indications in others. In 50% of the cases reported infants died within three to four weeks of the first sign of illness and diagnosis made at necropsy (Hudson, 1956).

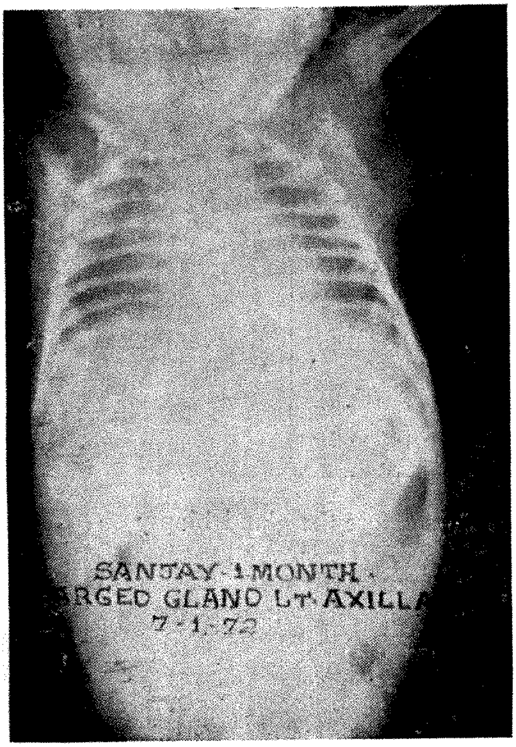

Fig. 1. X-ray of the infant at the age of one month, showing soft tissue swelling left axilla. otherwise not a clear plate.

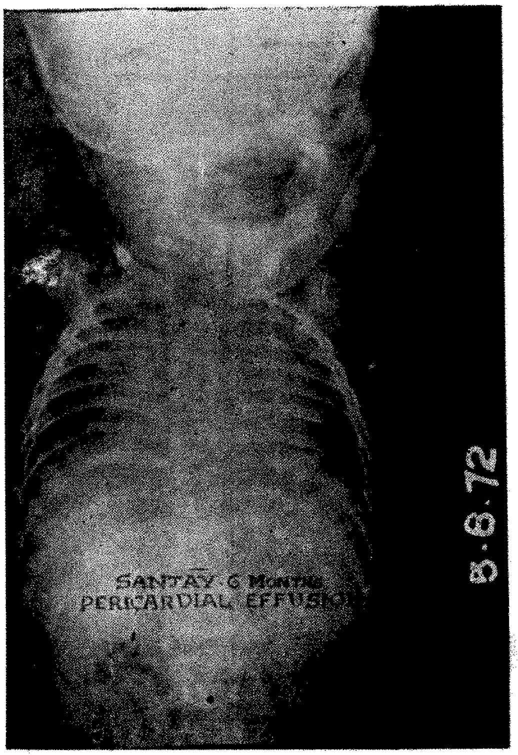

Fig. 2. X-ray chest at the age of six months, showing pericardial effusion.

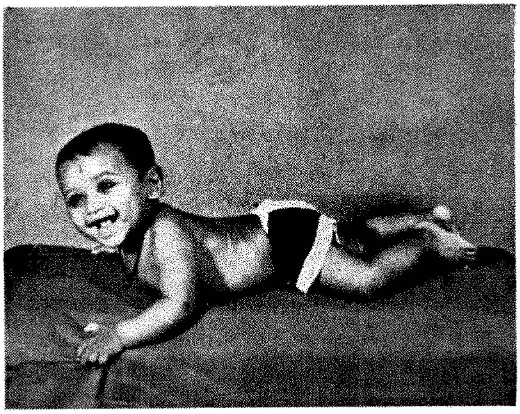

Fig. 3 photograph, of the infant two days before the gland biopsy

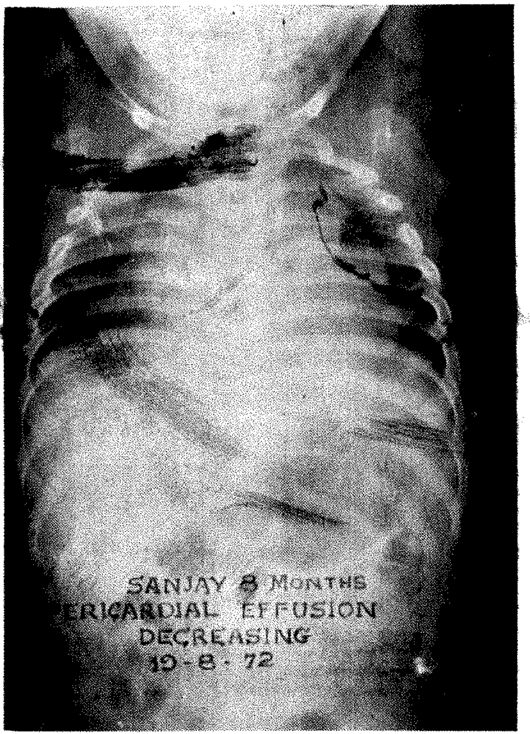

Fig. 4 X-Ray chest of the baby after two months treatment, Pericardial effusion less

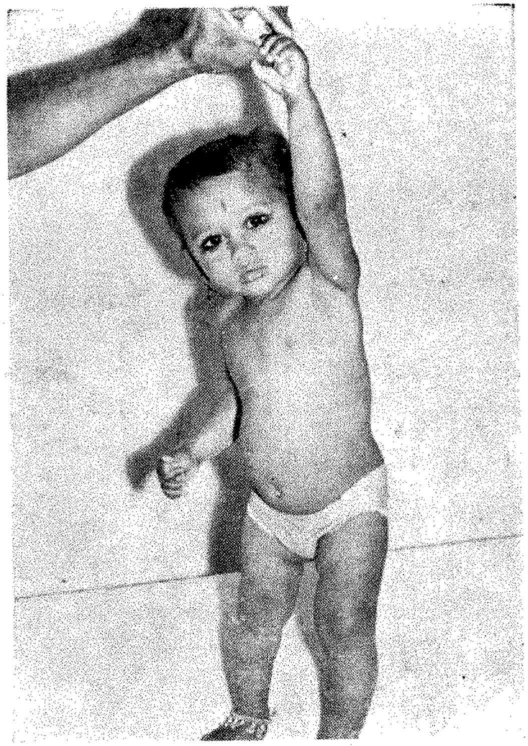

Fig. 5 Photogroph of the child, showing biopsy scar left axilla.

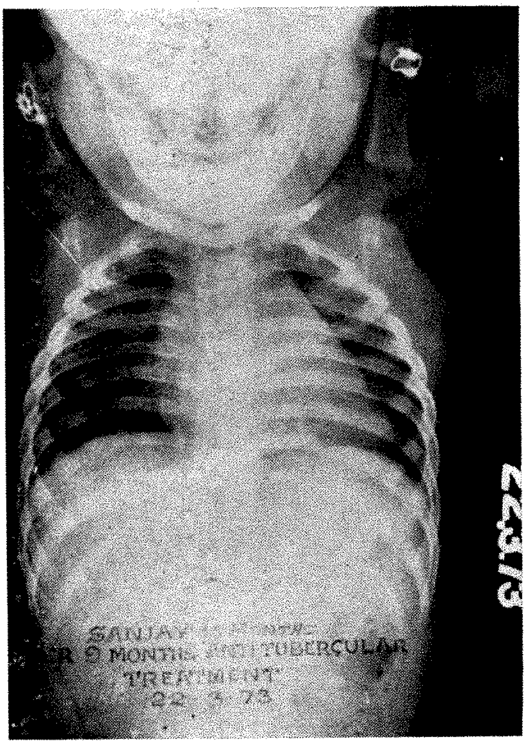

Fig. 6 Normal

Amick et al. (1950), reported two cases where presenting sign was enlargement of the cervical glands. No case has been reported with solitary axillary lymphadenitis or symptomless pericardial effusion.

The case reported is unusual because :—

(a) He had no other symptom except the mass in left axilla, (gland). He was apparently healthy and the growth was not affected.

(b) He had symptomless pericardial effusion, which responded to antitubercular treatment.

(c) Mother is infection free to date, and there is no history of tuberculosis in family or neighbourhood.

It sets one pondering what could be the source and mode of infection in this patient. We suggest that perphaps mother had tuberculous bacillaemia during the later months of pregnancy, and the bacilli lodged in the foetus instead of any tissue of the mother. BCG given four days after birth precipitated the infection contracted during intrauterine life. Due to the availability of antitubercular drugs child could be saved which was a remote possibility in Hughesdon's time (1946).

SUMMARY

A case of congenital tuberculosis is reported, who had solitary glandular enlargement in left axilla, with asymptomatic pericardial effusion. Mother has been infection free through out. Child is alive and growing normally.

Regarding the mode of infection in this infant it is suggested that, perhaps mother had tuberculous bacillaemia during pregnancy and the bacilli lodged in foetus instead of any organ or tissue of the mother.

ACKNOWLEDGEMENTS

We are grateful to Brig (now Maj Gen) N. Adiseshiah, then Commandant AMC Centre & School, Lucknow, for encouraging and allowing us to publish this case.

We are also thankful to the parents of the child for extending us full cooperation for the follow up.

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BUDD-CHIARI SYNDROME

(A Clinico-Pathological Study)

BY

Lt Col B.N. Chattoraj*

INTRODUCTION

In 1857 Budd first described this syndrome and reported three cases. In 1899 Chiari described the pathological changes and the condition came to be called Budd-Chiari Syndrome. He reported three cases of his own and collected seven others. This is an extremely rare condition. Armstrong and Carness (1944) found only five instances in 11,979 autopsies performed. Thompson (1947) reported two cases and could collect only ninety five cases from the literature.

By 1952, a total of 112 cases have been reported (Little & Montgomery, loc cit.).

Complete occlusion of the hepatic veins is extremely rare, and in most instances the obstruction is only partial and an incidental finding at autopsy. In a review of twenty cases of occlusion of hepatic veins, Kelsey and Comfort (1945), reported no corresponding, recognisable clinical signs in sixteen cases. Chiari (loc cit.) called the condition endophlebitis of hepatic veins, whereas, Thompson and Turnbull (1912) postulated thrombosis at the junction of the hepatic veins with the inferior vena cava. In most of the cases reported, there was an obliterating endophlebitis of the hepatic veins, starting in the larger radicles and often extending into the inferior vena cava. In some instances the smaller branches were affected. The condition is more commonly secondary to other diseases, such as invasion or pressure from adjacent neoplasms, vena caval spread of renal carcinoma, thrombophlebitis migrans or as a complication of renal neoplasms or polycythaemic states (Shoval, 1958).

The hepatic veins are usually obliterated or greatly narrowed as they approach the vena cava. Thrombosis may spread from the vena cava into the hepatic veins or even the ostia may be occluded completely. Primary occlusion of the hepatic veins in the liver substance beyond the vena cava is rare. Thrombus filling the veins may be purulent, or may contain malignant tissue, depending on the cause of occlusion (Sherlock, 1955).

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The clinical feature is of an ill patient suffering from some other condition, usually a malignant process and present with sudden vomiting, enlarged liver, ascites and mild jaundice. The symptomatology is the result of portal venous hypertension and hepatocellular inadequacy as a consequent of rise in pressure in the hepatic veins and sinusoids. Centrilobular destruction of liver cells results in parenchymal damage. Vomiting and pain over the liver, from distension of the hepatic capsule, and diffuse watery diarrhoae following mesenteric vein thrombosis are other features. The liver is enlarged and tender. The ascites is gross and is resistant to all forms of treatment. This syndrome can also occur in a chronic form with gradual onset, vague indigestion, hepatomegaly and ascites. Acute episodes develop following thrombosis of hepatic veins, which have only been partially occluded. All features of chronic portal hypertension develop in a few months and eventually these patients die usually of hepatic failure.

Grossly, the liver appears purplish, smooth and enlarged. Venous congestion is gross and the cut surface shows an advanced "Nutmeg" appearance. Prominence of hepatic veins is apparent. The spleen may be enlarged if the patient lives long enough. Microscopically, centrizonal necrosis, dilatation, sinusoidal congestion and haemorrhage, with necrosis of the liver cells and dilated lymphatics are seen. Endophlebitis obliterans and thrombi in various stages of organisation are present in the radicles of the main hepatic veins. In more chronic cases there is replacement fibrosis and the picture of cardiac cirrhosis.

CASE HISTORY

This patient aged twenty four years was admitted with the complaints of abdominal pain, headache, giddiness of about two months duration. He had frequent vomiting ten days prior to admission and had been treated a year back for amoebic hepatitis. On examination there was no anaemia, no fever and nutrition was fare. Ascites with hepatomegaly and prominent superficial abdominal veins were evident. There was no jaundice. While in hospital he became comatose but recovered later. He was treated as a case of cirrhosis with hepatic precoma. His condition became progressively worse. Ascites and hepatomegaly with somewhat firm nodular liver persisted. He developed jaundice and oedema feet; and had episodes of haematemesis and mild albuminuria. Blood examination revealed a normocytic anaemia. He was treated with cortisone, antibiotics, blood transfusion and other supportive measures. He died on the fifth day as a result of hepatic coma.

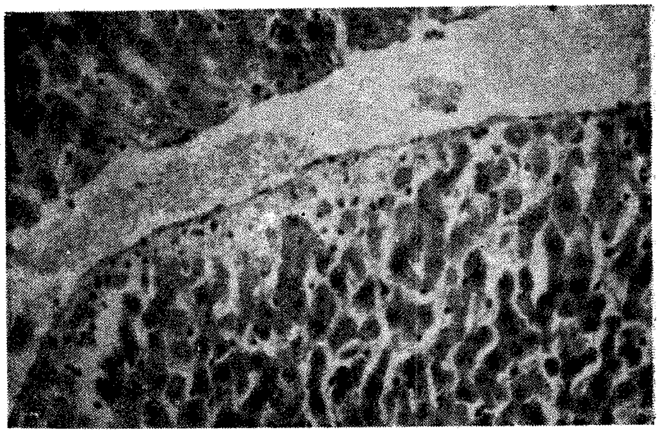

Fig. 1 H E of the Liver shows a large radicle of the hepatic vein containing well organised thrombi. Part of the the thrombus has been washey away during the preparation of the histological specimen.

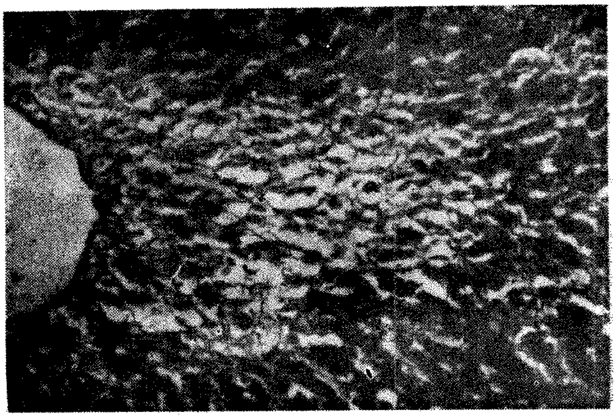

Fig. 2 H E of the Liver shows necrosis of the centrizonal parencbymal calls.

POST MORTEM EXAMINATION

A young emaciated adult male with moderate icterus of the conjunctiva, protuberant abdomen and slight oedema of the back and ankles. There was no lymphadenopathy, clubbing of fingers or purpuric spots. Umbilicus was somewhat everted. No scrotal oedema was seen.

Alimentary System:—Veins in the lower half of the oesophagus especially near the cardiac end showed moderate varicosity. Stomach appeared normal. There was altered blood mixed with mucus in the lower ileum and the rest of the gut as fas as the transverse colon. No ulcer or tumour was seen anywhere in the gastro-intestinal tract.

Liver:—Was fairly noduler, weighed 1800 gm, cut with resistance and had the appearance of a gross long standing passive venous congestion, not unlike cardiac cirrhosis. The cut surface had a somewhat purple colour mottled with streaks of grey. In the larger venous channels there were well organised occluding thrombi. The gall bladder contained 20ml of normal bile. No gross tumour was identified. Spleen was firm, enlarged (340 gm.), with prominent trabecular markings and congested pulp. The peritoneal cavity contained five litres of clear icteric fluid. Other organs showed mild to moderate congestion.

HISTOPATHOLOGICAL EXAMINATION REPORT

The hepatic architecture was disrupted by extreme venous congestion and superadded early cirrhotic changes. The larger radicles of the hepatic vein showed well organised thrombi (Fig 1) with recanalisation at places and microscopic areas of mineralisation. There was marked focal and periportal round cell infiltration along with collapse and necrosis of the centrizonal parenchymal cells (Fig 2). Patchy microscopic areas of haemorrhage were seen extensively. Bile duct proliferation was seen to a slight extent. There was no bile retention or tumour.

Spleen, kidneys and intestines showed evidence of chronic venous congestion.

DIAGNOSIS : Chronic Hepatic Vein Thrombosis (Budd - Chiari Syndrome)

DISCUSSION

The gross appearance of the liver and the rest of the viscera were characteristically that of a Budd - Chiari Syndrome. The actual cause of hepatic vein thrombosis in the case presentend is not apparent in the autopsy study, grossly, as the superior vena cava below the liver was not showing any thrombus. Nor was there any evidence of any tumour macroscopically or microscopically. Possibility of an endophlebitis can be considered as the etiological factor, as evident from the inflammatory process in the liver beside the thrombi. Cases with this syndrome usually end fatally within some weeks but some cases may live for many months. In this case the fatal illness must be presumed to have started a year back. Hence the course of the present illness could be deduced to be about seven months. All the symptomatology resulted from the portal venous hypertension and hepatic cellular inadequacy. The mild cirrhotic change was secondary to the hepatic venous thrombosis and consequent venous congestion. The histological changes revealed a long continued process with well organised hepatic venous thrombi and a chronic endophlebitis. From the clinical, post mortem and histopathological findings it is obvious that this was a case of Budd–Chiari Syndrome, who died of hepatocellular inadequacy.

SUMMARY

The condition of Budd – Chiari Syndrome has been briefly reviewed. The etiology, clinical features and pathology of the condition have been discussed.

An extremely rare case of Budd – Chiari Syndrome has been described.

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STRESS FRACTURES OF TIBIA

(A study of cases occuring at Madras Regimental Centre)

BY

Lt Col B.N. Ghosh* and Maj Bhaskaran**

INTRODUCTION

Fatigue or stress fracture is a familiar condition at regimental centres where physical training of a large body of troops takes place regularly. Lack of consciousness of the condition results in delay in diagnosis with consequent prolonged incapacitation and hospitalisation. Cases of stress fracture occuring in recruits of the Madras Regimental Centre during the period September 1967 to April 1971 are being reported here.

Incidence

A total of twenty six cases have been detected during this period; breakdown for each year is shown in Table I.

Table I

Incidence of Stress Fracture of Tibia at M.R.C.

Period	Total No. of Recruits (Average)	Total No. of cases	Incidence per thousand
1967 (Aug. -Dec.)	–	5	–
1968	1010	4	4.0
1969	980	6	6.1
1970	800	9	11.2
1971 (Jan. - Apr.)	–	2	–

A higher incidence in 1970 is probably due to more consciousness of the condition on the part of R.M.O. and thorough screening.

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** R.M.O., Madras Regimental Centre, Wellington, at present with 23 Mtn Div.

Age

All patients were in the age group of 18 - 20 years, evidently as this is the age of recruit entry and their training period. It was not detected in any of the trained soldiers.

Diet

All patients came from lower Socio-economic Strata. Seventeen patients were vegetarian and nine were nonvegetarian. No increased incidence was detected in any of the two groups.

History of Trauma

All patients except two gave a definite history of trauma. Different types of trauma encountered are shown in Table II.

Table II

Types of Trauma

Event	No. of Patients	Percentage
Fall	15	57.7
Jumping	5	19.2
Route march	2	7.7
Direct injury	2	7.7
No history of injury	2	7.7

A history of injury was detected in all cases except two. Commonest type of injury was fall during running, games, obstacles and bayonet training (57.7%). Next common cause of injury was jumping over P.T. horse, trench and high jump (19.2%)

Duration

Patients gave history of injury dating one day to one month back, commonest period was 10 days. Most of the patients sustained injury in the morning time (0800 - 1100 hours) probably as the recruit training is at its peak at this period.

Presenting symptoms

Commonest presenting symptom was pain, next was swelling or protruberance over upper one-third of tibia medially.

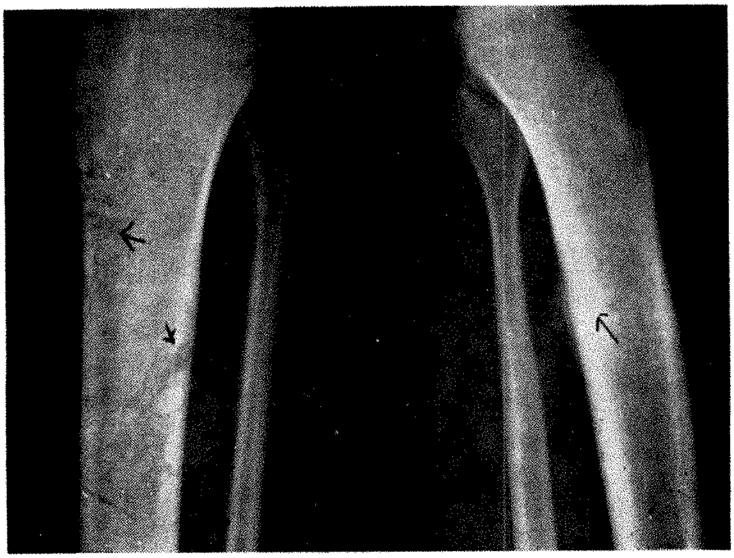

Fig. 1 Stress fracture upper – third tibia showing sub – cortical callus formation.

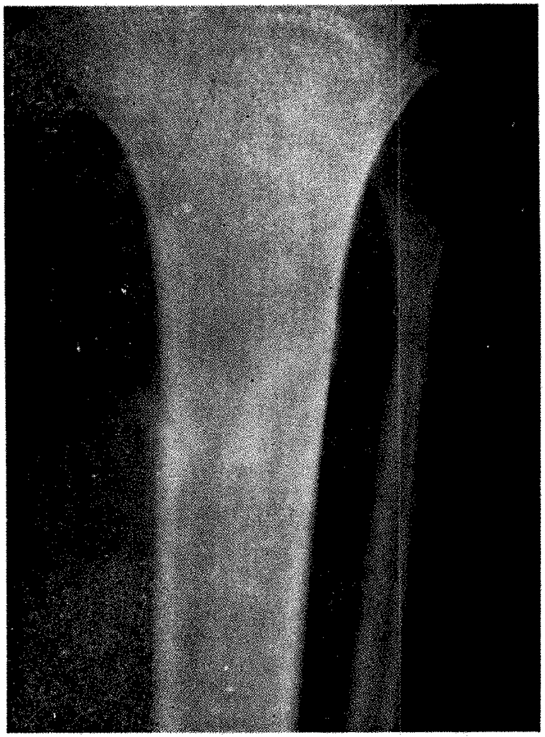

Fig. 2 Linear crack fracture of tibia in association with stress fracture. (A. P & Lat).

Associated anomalies

No associated congenital anomalies e.g., unequal leg length, genu valgus or varus were detected. Bilateral hallux was seen in one patient.

Site of injury

It is striking that in all cases upper-third of tibia was the site of fracture. Right side was involved in 14 cases and left in 12.

Laboratory Investigations

Haemoglobin percentage, total serum protein and serum calcium were within normal limits. No abnormality was detected in urine examination. WR., Kahn and V.D.R.L. were negative in all cases. (to exclude syphilitic periostitis).

X-Ray

Most cases showed subcortical callus formation postero-laterally. (Fig. 1).

Treatment

Patients were divided into two groups for purposes of treatment. Group A : (12 patients) treated by P.O.P. immobilisation for 3-4 weeks. Walking heel/caliper applied after a week. Physiotherapy was instituted for 1-3 weeks after removal of P.O.P. These were the patients with severe pain and tenderness and were unable to bear weight or walk.

Group B : (14 patients) treated with bed rest and early physiotherapy. These patients required hospitalisation for an average period of three weeks. It took another 2-4 weeks before they were fit for all duties. Two patients in whom stress fracture was not recognised earlier were admitted later with complete crack fracture of tibia. (Fig. 2).

Follow up

Cases were followed up from 3 months to 2 years. Most of the patients whose follow up period were less than 1 year recovered completely and were posted out from the centre. Prominence at fracture site with pain and tenderness continued for some unexplained reasons in 5 patients for long periods (4-6 months) although radiologically union was complete with good consolidation. Other complications noted were, stiffness of knee (3 patients), sprain knee (one patient) and complete fracture tibia (two patients).

Only two patients required down-grading to lower medical category (B or C) over a year.

DISCUSSION

Fatigue or stress fracture is now regarded as a "process" where alteration of bones results from stress. A fracture may or may not develop and if it does, it can be considered as a complication of the stress reaction (Loren and Morris, 1966). The precipitating traumatic incidents are usually mild and every day incidents like running, jumping and marching, indeed their exact nature cannot often be determined (Murrey and Colwill, 1968). Rarely can these incidents be regarded as sufficiently severe to have produced a frank fracture, it appears reasonable, therefore to regard a defect in bone structure as a predisposing factor. Although frank evidence of malnutution was not detected biochemically during the present period of investigation it is possible that inadequate nutrition during their growth period led to a defective laying of bone lamellae (as all patients pertained to lower socio-economic strate). This bone (cortex) is unable to with-stand the repeated stress and results in a subcortical crack fracture finally. Though fatigue fracture of metatarsal is classically described after a long route-march (Watson-Jones, 1962), it was not detected in any of our patients. The site was upper third of tibia in all cases. This appears related to the type of training so that stress primarily falls on a particular bone – in our cases upper-third of tibia. Stress fractures of femoral neck, fibula, calcaneus (Loren and Morris, 1966), tarsal navicular (Lorris et al., 1970) and pars interarticularis (Murrey and Colwill, 1968) as described by some authors were not seen in our series. It is essential to detect these cases early in order to save 'manhours' lost and is equally essential to give these patients complete bed-rest or protection of a splint or P.O.P. cast, otherwise there is a danger of some of them going on to complete fracture of shaft of tibia.

SUMMARY

Twenty-six cases of stress fracture of tibia are described. Their aetiological factors and treatment have been discussed.

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CALCINOSIS UNIVERSALIS AND DIABETES MELLITUS IN SCLERODERMA

(A Case Report)

BY

Wg Cdr P.N. Bhargava* Lt Col M.K. Ghosh Sqn Ldr S.K. Dham*****
Lt Col N.R. Krishnan** Surg Capt G. Kuppuswamy, VSM†**

It is generally believed that the cutaneous lesions of scleroderma are merely the outward manifestations of a generalised disease affecting the muscles, bones, heart, lungs and gastro-intestinal tract [9,6]. The disease usually progresses relentlessly and death results from progressive involvement of the cardio pulmonary system or with rapid involvement of the kidneys[6].

In some cases of scleroderma, calcification is seen in palmar aspects of the terminal phalanges as dense nodules and in the form of irregular masses or plaques in subcutaneous tissues, particularly in areas subject to pressure, such as gluteal regions, lower back and shoulders. Wide spread calcification or calcinosis universalis, (diffuse calcinosis), in scleroderma is rare.

We report a case in whom the incidental discovery of extensive subcutaneous calcification led to the final diagnosis of scleroderma.

CASE REPORT

Smt. Mariappan, aged 36 years, was referred for an X-ray of the right knee. She had sustained an injury to the knee due to a fall two months previously in July, 1971. She was almost bedridden. An X-ray of the right knee revealed recent fracture of lower end of femur undergoing union. Extensive calcification was also noted incidentally in the subcutaneous tissues. The patient was admitted to this hospital for detailed investigations. On admission, patient complained of: progressive general weakness and inability to move about due to restriction in movements of both knees, of six months duration. She did not give any history of recent fever, chest pain, difficulty in swallowing, paraesthesias or history suggestive of Raynaud's phenomenon. She had been having breathlessness on accustomed exertion and cough off and on for past six years. The cough was not of a productive nature and she never had any haemoptysis.

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Past History

The patient had been treated as a case of pulmonary tuberculosis in 1965–66. No history of any peptic ulcer like illness.

On Examination

A thinly build and emaciated individual. Marked bilateral parotid gland enlargement present. BP 130/80 mm Hg. Afebrile. Skin: Glossy. Almost adherent to the deeper tissues over neck, upper and lower limbs. The fingers were thin and tapering, showing some depigmentation over the tips. Nails atrophic. Skin over the anterior aspect of thighs was scarred. Hard irregular granular and nodular masses felt extensively under the skin. No abnormal pigmentation or telangiectasia was noted.

Joints

All the joints and interphalangeal joints showed marked restriction of movements; however, there was no significant deformity or swelling of the joints.

Systemic Exam

CVS–apex beat 5th interspace within midclavicular line. No parasternal heave was present.

2nd Pulmonary sound–loud and split. A Grade III systolic murmur over the apex not conducted to axilla.

Lungs-generalised diminution in the air entry.

No adventitious sounds were detected.

Abdomen–distended with no evidence of free fluid.

No hepatosplenomegaly.

CNS–no neurological deficit detected.

Investigations

Hb 13.0 gms%. ESR 48 mm fall at the end of 1st hour. TWBC 7,900/cmm. DLC - P 40, L 57, M 1, E 2%. PVC 40%. MVC 97.5 cm. MCH 35 rr G. MCHC 36.25%.

Urine (Routine Exam) revealed presence of sugar, (yellow reduction with Benedict's), trace of albumin and occasional pus and epithelial cells. Urine culture was sterile. Blood Urea - 35 mgm%.

G.T.T.

Time	0	½	1	2	hrs
Blood Sugar	183	316	333	308	
Urine Sugar	Green	Brick red	Brick red	Brick red	

Electrophoresis of Serum Proteins :—

Total proteins 7.0 gms%. Albumin 42% of total proteins. Alpha 1 - 12.1% of total proteins. Alpha 2 - 14.2% of total proteins. Beta - 6.7% of total proteins. Gamma - 25% of total proteins.

Serum calcium	: 10.0 mgm% (Average)	Done on three occasions.
Serum Inorganic Phosphate	: 3.4 mgm% (Average)	

Serum sodium : 135 m Eq/litre. Serum potassium : 4.3 m Eq/litre. LE cells in blood - not detected.

Muscle Biopsy (Microscopic Examination)

Homogenisation, sclerosis and atrophy, of striated muscle bundles with increase of interstitial connective tissues. Vessels showed obliterative changes. Picture consistent with systemic scleroderma.

X-ray Examinations

Chest PA - Small patchy opacities were present in both upper and midzones and left basal region. Main pulmonary vascular shadows were enlarged. The peripheral vessels showed elongation, tortuosity and abrupt termination. The heart and aortic outline was shaggy.

Cardio : thoracic ratio - 13 : 25 cm.

Osteoporosis affecting the upper end of humerus on both sides was present. Multiple small irregular, coarse nodular and plaque-like areas of calcification were seen in the soft tissues of the upper arm, thorax and breasts.

Abdomen (Fig 1) - Kidney shadows are rather large for the build of the patient, and dense. No evidence of urinary calculi or calcinosis. Extensive, irregular plaque-like and nodular areas of calcification seen in the region of the buttocks. The transverse processes of lumbar vertebrae and bones of the pelvis show slight generalised osteoporosis.

Hand and Wrists (Fig 2) -Moderate generalised osteoporosis is present. Diminution of soft tissue shadows is seen. Fingers are tapering. There is loss of joints spaces at the interphalangeal joints and at the wrist. No calcification seen in the soft tissues of the digits.

Hip Joints and Thighs (Figs 3 & 4)- Extensive irregular, nodular calcified opacities are seen in the soft tissues on both sides. Plaqes like calcification is seen on the lateral aspect of the thigh. There is lack of musculo-fascial detail in the soft tissues. The femoral artery is calcified on both sides.

Knee Joints (Fig 5) - Old, well united fracture of lateral condyle of femur (Rt). Slight genuvarus deformity present on both sides, more marked on the right. Multiple small calcification seen in the soft tissues.

Barium Swallow Examination - Poor peristalsis and slow evacuation was noted in the oesophagus. Tertiary, ripple like peristaltic waves also seen in the "spot" radiograph. There was slight narrowing in the middle third with persistent slight dilatation above the narrowed segment. No evidence of oesophageal reflux or peptic oesophagitis seen.

Follow-up examination did not reveal any significant abnormality.

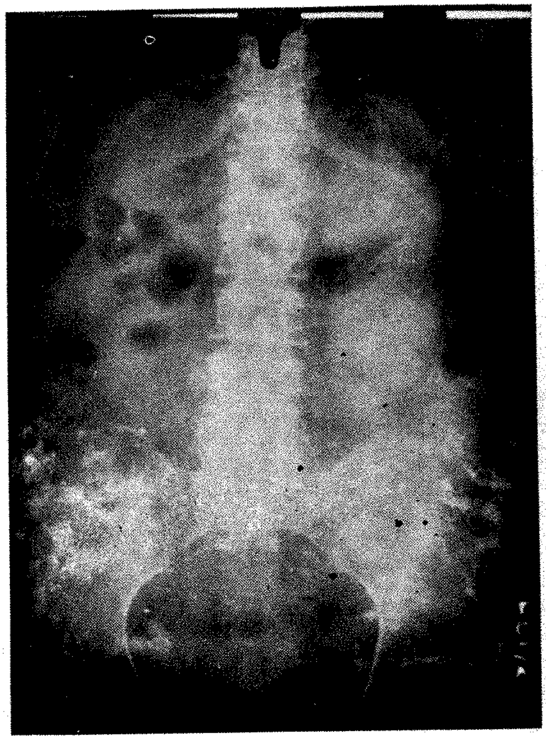
X-ray Skull - did not reveal any significant abnormality except for slight osteoporosis.

E C G (Resting)

Within normal limits.

Pulmonary Functior Tests

Revealed marked diminution in vital capacity, total lung capacity and resting ventilation, and moderate decrease in maximum expiratory flow rate consistent with a restrictive and obstructive defect.



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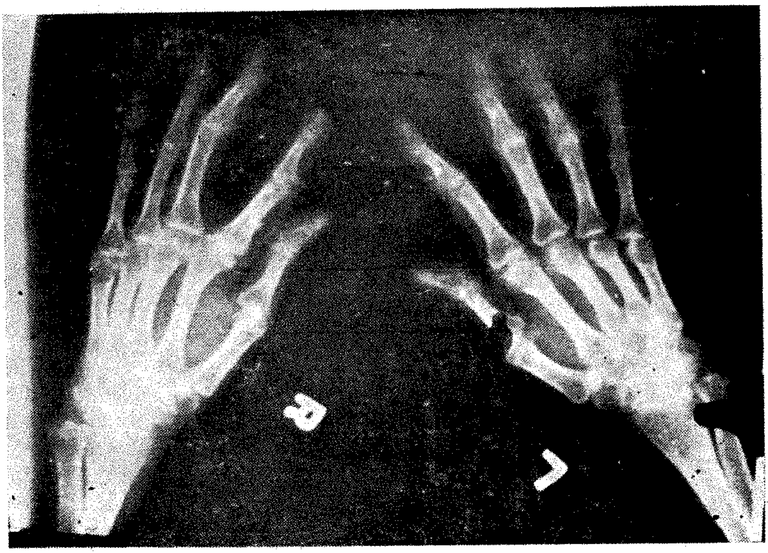

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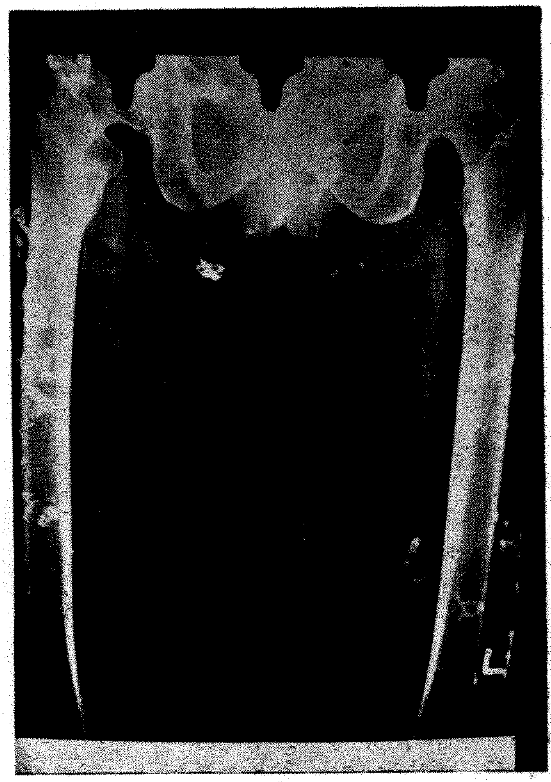

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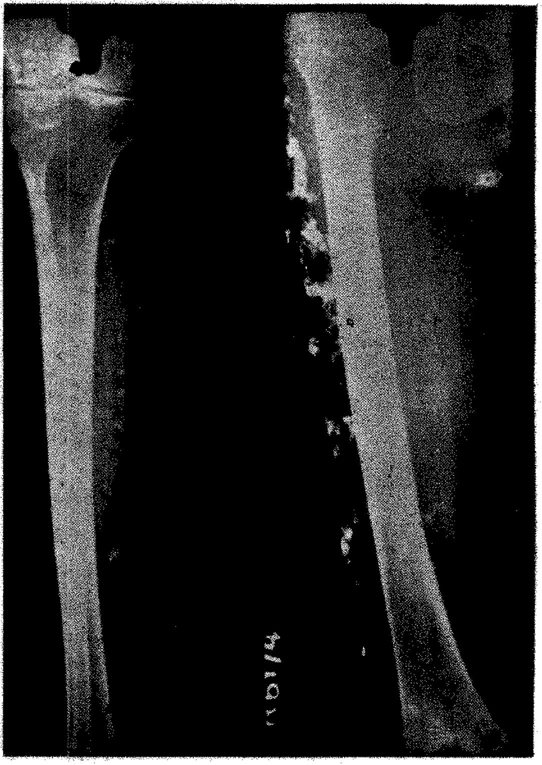

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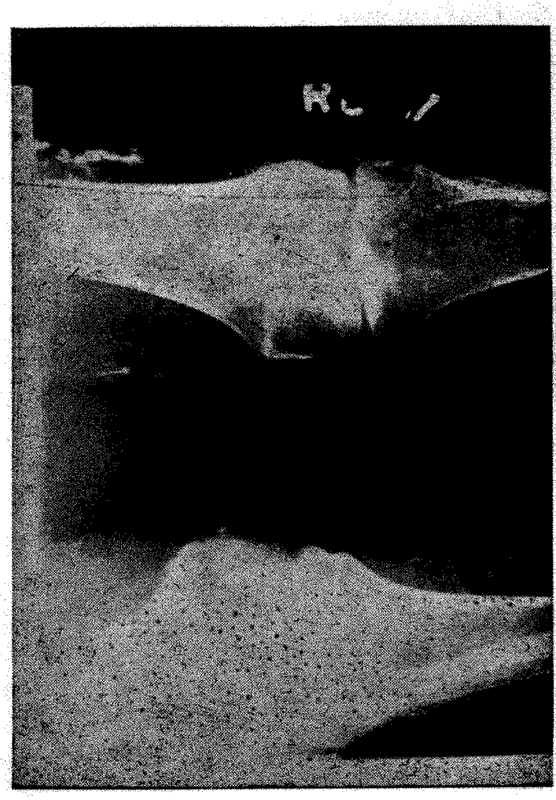

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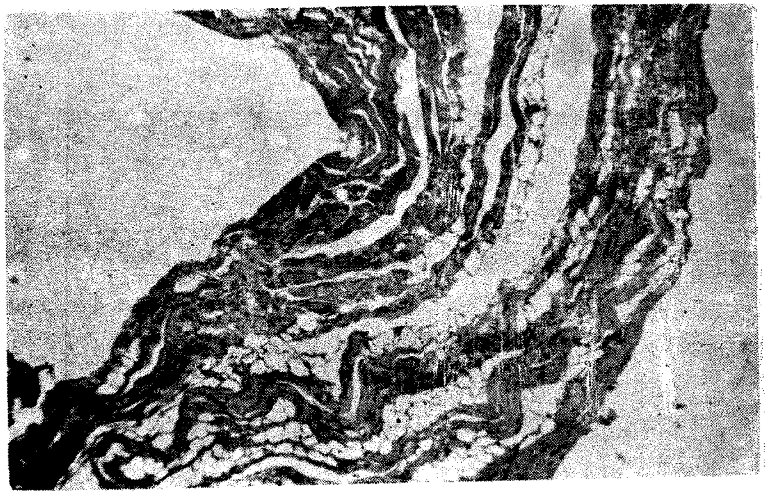

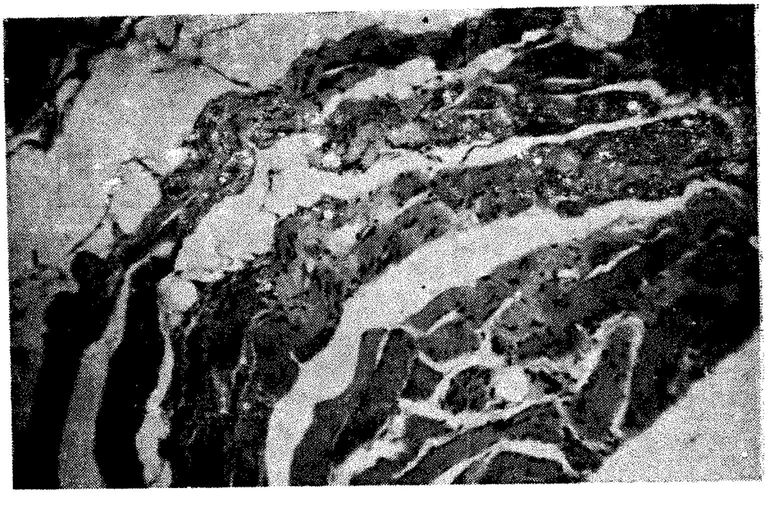

DISCUSSION

Sclerosis of the skin and muscles with Raynaud's phenomenon in the distal phalanges of fingers and calcinosis are diagnostic of scleroderma.

Exceptionally the skin escapes and the lesions are confined to the deep viscera. According to Kerley[10] in at least 50 percent of cases the pulmonary lesions precede the skin lesions and are overlooked or wrongly identified. The lung lesions present as a diffuse interstitial fibrosis of the linear nodular type. The pattern of X-Ray appearances of the lung in the presented case is not typical of old tuberculosis and it now can be presumed that the lungs were involved in 1965-66 when the patient was treated as a case of pulmonary tuberculosis.

In many of the advanced cases, cardiac enlargement is present. This may be due to cor pulmonale, cardiac ischaemia or pericarditis.

Marked dimunition of pulmonary functions indicates more extensive fibrosis than revealed by chest radiograph. Guleria [2] et al (1970), also emphasise the greater reliability of pulmonary function tests in assessing the impairment of respiratory function rather than the gross X-Ray appearances of the lung. Kemp Harper [3] (1965) and Frazer[8](1966) consider radiology the best method of assessing the extent of viscreral involvement. Subcutaneous calcinosis was present in 58 and 50 percent of their cases respectively. In Fraser's [8] series, soft tissue calcification was encountered mainly in the hands which varied from widespread chunky calcification to tiny discrete flecks seen in the tips of the fingers. In the present case soft tissue calcification is not seen at all in the hands but is marked in the region of the buttocks, thighs, legs, breasts and shoulder region in that order. Femoral artery calcification was seen on both sides.

Other conditions such as hyperparathyroidism, Vit D intoxication and milk Alkalic syndrome were excluded by absence of hypercalcemia and a negative history of either having taken heavy doses of Vit D or of any illness suggestive of peptic ulcer when she could have been given excessive quantities of milk and alkalies. In Ehlers-Danlos Syndrome, widely disseminated rounded opacities in the subcutaneous tissues of the limbs occurs due to calcifications in lobules of fat. Absence of hyperelasticity of skln, hypermobility of joints and capillary fragility, preclude the possibility of Ehlers-Danlos Syndrome.

Association of Diabetes Mellitus with Scleroderma is well known. Since glucose tolerance tests are not routinely done, and invariable diabetes mellitus in all cases of scleroderma cannot be predicted in every case.

So far, scleroderma has proved resistant to all therapy. Steroids, paraminobenzoic acid, low molecular weight dextran[4] and progesteron[1] have all been tried. Aluminium hydroxide gel proved useful in treatment of calcinosis due to Dermatomyositis in one case[11]. The present case was treated with small doses of steroids and aluminium hydroxide gel without any encouraging results.

SUMMARY

A case of extensive subcutaneous calcification in a case of scleroderma is presented. She had associated moderately severe Diabetes Mellitus. It is recommended that progressive systemic sclerosis be considered in every case presenting with soft tissue calcification and tests of glucose tolerance performed to exclude Diabetes Mellitus in such cases.

ACKNOWLEDGEMENT

The authors express their gratitude to the Commanding Officer, Air Force Hospital, Bangalore, Group Captain P Basu, for his kind permission to publish the case report. We are also grateful to Sqn Ldr N Ramachandran for the photographs of this article.

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FRACTURE DISLOCATION OF THE PELVIS

(A Case Report)

BY

Lt Col C.S. Panu*

Pelvic ring is formed by articulation of hip bones with the sacrum posteriorly and with each other anteriorly at the symphysis pubis. Anterior pubic segement of the pelvic ring provides protection to viscera where as remaining postero-lateral iliac segment serve to support and transmit the body weight.

Fracture and dislocation of the pelvis may be caused by muscular pull, direct or indirect violence/strain at any age irrespective of sex.

An athlete whose epiphysis are not united when faced with sudden uncontrolled efforts during games like foot ball may detach any of the following :—

(a) Anterior inferior iliac spine (Avulsion of the rectus femoris).
(b) Anterior superior iliac spine (Avulsion of the sartorius).
(c) Epiphysis of ischium (Avulsion of the hamstrings).

These are simple injuries and a few weeks rest in bed is sufficient to cure the injuries.

Direct localised closely circumscribed violence causes isolated injury of the pelvis like fracture of the sacrum, fracture of the coccyx, fracture of the ischium or fracture of the ilium. In these cases a few weeks recumbency is required to relieve the pain.

Indirect violence to the pelvis in the form of compression from front, from sides or obliquely which is sustained when an individual is buried, run over by a vehicle or due to fall from a height may cause single or a combination of injuries like :—

(a) Unilateral fracture of one or of both pubic rami
(b) Bilateral fracture of one or of both pubic rami

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(c) Unilateral fracture of both rami with separation of symphysis pubis.

These injuries do not cause any deformity, shortening, or alteration in the alignment of weight bearing joints except local tenderness. A few weeks recumbency is essential for recovery.

(d) Dislocation of the symphysis pubis with a dislocation of the sacroiliac joint.

(e) Less frequently (i) dislocation of the symphysis with a fracture of the ilium near the sacroiliac joint (ii) a fracture of both pubic rami with a dislocation of scroiliac joint. In such cases one half of the pelvic girdle is widely displaced carrying with it the lower limb so that there is a deformity and shortening.

(Reference is made to term of Malgaigne fracture. When pelvic girdle breaks in front and behind it is termed malgaigne vertical fracture. When there is a fracture of both pubic rami on one side and Fracture of ilium or massa lateralis of the sacrum on the opposite side it is termed crossed Fracture of Malgaigne).

Such cases are treated by reduction by manipulation in lateral recumbeney on sound side and immobilisation in double hip spica for 12 weeks.

In such cases the lateral fragment with hip and whole limb way lie 1-3 cms higher than the sound side. It may be rotated outwards and displaced towards the midline. The posterior superior iliac spine becomes prominent and is nearer the midline. The pull of muscles which pass from the medial fragment to the thigh and from the trunk to the lateral fragment maintain the displacement. By traction in the long axis of the abducted limb this displacement may be corrected and reduced position is maintained by continuous traction.

The displaced fragments of the pubic bone may cause injury to the urethra, urinary bladder, rectum or vagina. Strangulation of a coil of small intestine in the displaced sacroiliac joint can occur. Nerve injuries (obturator nerve/sciatic nerve) are not uncommon. Injuries to large vessels are rare but extensive haematoma formation may be noticed. So during examination patient may be asked to pass urine or be catheterized. P R should never be omitted.

In a case of Fracture Dislocation of the pelvis presented below an unusual combination of injuries to the pelvic girdle was detected.

A Sepoy aged 22 years was involved in 1 Ton Lorry accident on the 24th August 1965. (Vehicle fell into a khud). He was occupying the front seat. He did not remem-

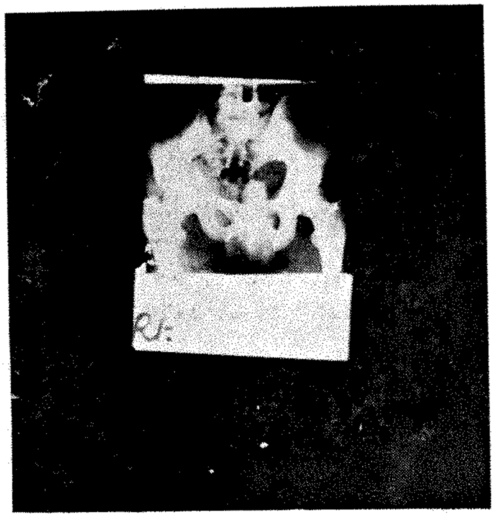
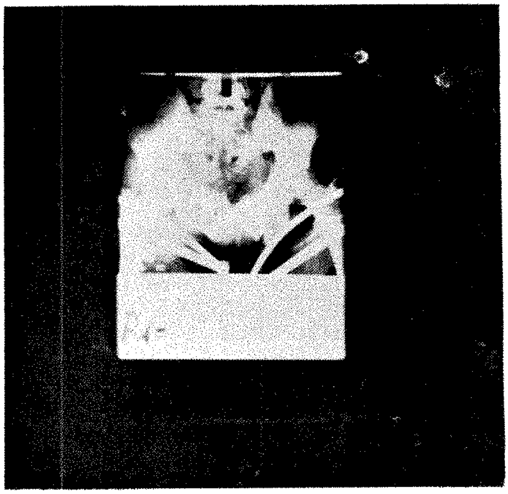


Fig. 1



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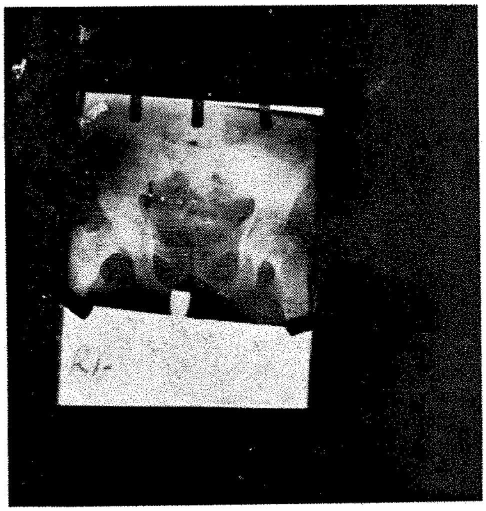

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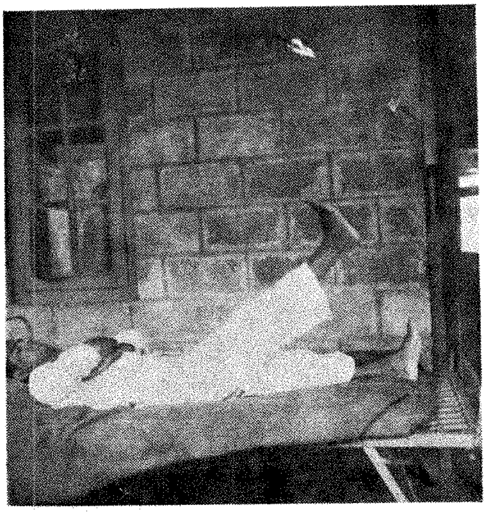

Fig. 4

ber what happened to him but had a faint idea that his vehicle went off the road on that day. In that vehicle accident he sustained the following injuries :—

(a) Contused wound scalp.

(b) Contused wound upper 1/3 of the thigh Lt.

(c) Injuries to the pelvic girdle (Fig. 1)

  (i) Fracture Sacrum
  (ii) Fracture both pubic rami Lt
  (iii) Dislocation of symphysis pubis
  (iv) Sublaxation of the sacroiliac joint Lt.
  (v) Central dislocation of the hip joint Rt.

In Rt lateral recumbency POP double hip spica was applied on the 25th August 1965. It was removed on the 29th Sep 1965. Distance between the mid point of the symphysis pubis and the greater trochanter was 1/2 an inch short on the Rt side. Movements of the Rt hip were painful and restricted.

Fixed skin traction to both lower extremities was applied on the 29 Sep 1965. Check X-ray of the pelvis after 48 hours (Fig. 2) revealed that central dislocation of the Rt hip was reduced. Floor of the acetabulum was reformed. Sublaxation of the Lt sacroiliac joint was reduced. Traction was removed on 17 Nov 1965 and check X-ray (Fig. 3) revealed that reduced position of the Rt hip and of the Rt sacroiliac joint was maintained. Distance between the mid point of the symphysis pubis and the greater trochanter was equal on both sides. Active movements in bed were permitted. (Fig. 4). Photo taken on 24 Nov 1965, revealed that he could raise the Rt lower extremity by 60° comfortably (7 days after active movements in the bed).

DISCUSSION

Mechanism of above injuries to the pelvic girdle he could not state but as judged from above injuries it was presumed that the pelvic girdle was subjected to direct and indirect violence.

Fracture of the sacrum and of both pubic rami did not require any special treatment but a few weeks recumbency was good enough for recovery.

Dislocation of the symphysis pubis is reduced by pressure on either side of the symphysis and the reduced position is maintained by suspension in a pelvic sling. This procedure could not be applied as presure of the pelvic sling on the Rt greater trochanter might have prevented the head of the Rt femur being pulled out.

Subluxation of the sacroiliac joint is reduced by manipulation in lateral recumbency on the sound side and immobilisation in P O P double hip spica. (He was already in double hip spica when the case was taken over). This procedure was not to be applied in this case as there was no sound side. Rt lateral recumbency puts pressure on the greater trochanter and prevents reduction of the central dislocation of the Rt hip joint.

By continuous fixed traction to the Rt lower extremity, head of the Rt femur was pulled out, floor of the acetabulum was reformed and this reduced position of the hip joint was maintained. Similarly sublaxation of the Lt sacroiliac joint was reudced and reduced position was maintained by continuous fixed traction to the Lt lower extremity.

SUMMARY

A sepoy was involved in a vehicle accident and sustained an unusual combination of injuries to the pelvic girdle. Continuous fixed skin traction was applied to both lower extremeties. It reduced and maintained the reduced position of central dislocation of the Rt hip joint and subluxation of the left sacroiliac joint. After its removel, active exercises and physio therapy made him walk about comfortably and movements at the respective joints were painless.

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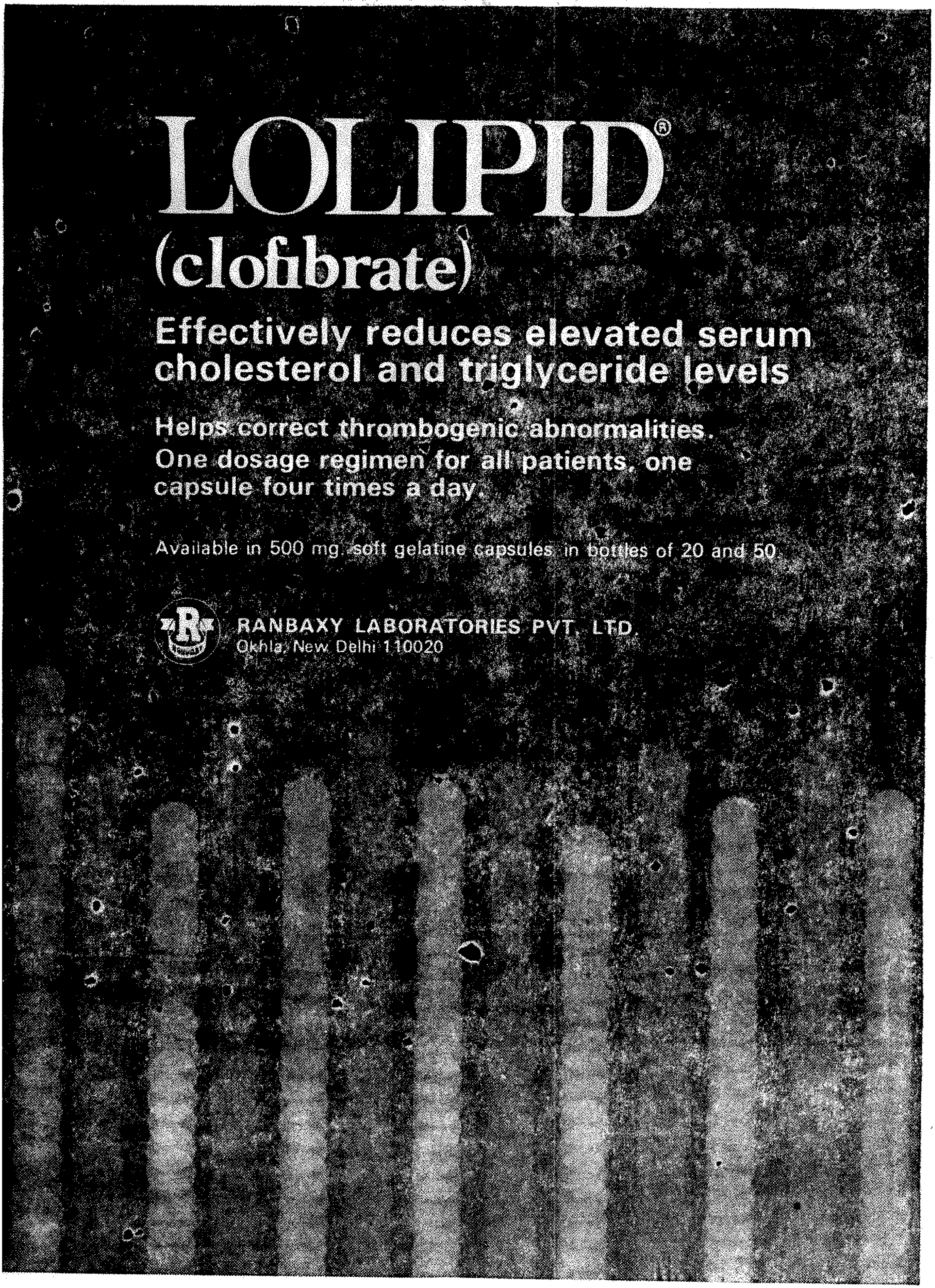

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The layout of the article, should in general, conform to the following format (a) Introductory paragraph (b) Material and Methods (c) Results (d) Discussion (e) Summary (f) Acknowledgement (if any) and (g) References. Spellings should conform to current English usage according to the Oxford Dictionary. Tables should have appropriate short headings, containing summaries of observations and relevant statistical data. Photographs, microphotographs and positive prints of radiographs should be printed on glossy paper. They should be submitted unmounted and should in no case be punched in the process of despatch. Each illustration should be numbered at the back and the legend referring to it should be typed on a separate paper.

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DIAGNOSTIC CHALLENGE OF RHEUMATIC ACTIVITY IN PATIENTS WITH RHEUMATIC HEART DISEASE

BY

Dr. Sujoy B. Roy*

In the early forties during the second World War, two Army General Hospitals from the same locality in the United States sent rather unusual medical returns. One hospital indicated that acute rheumatic fever was a common disease whereas the neighbouring hospital found it to be extremely rare. The reason for this discrepancy was due to the fact that more liberal army physicians from the first hospital equated rheumatic fever with sore throat, whereas the more conservative physicians of the second hospital demanded strict criteria including death from fatal rheumatic fever and necropsy evidence of aschoff bodies.

Under these perplexities the army authorities approached the late Dr. T. Duckett Jones who was then the Research Director of House of Good Samaritan at Boston, USA. This hospital for the preceding 20 years had treated acute rheumatic fever and established valvular heart disease in the children below the age of 17 years. Duckett Jones communicated his experiences that had guided him in the diagnosis of rheumatic fever in the Journal of American Medical Association in 1944. He outlined certain common features (Table 1) as he had observed occuring in rheumatic fever and tabulated them as major and minor criteria. A decade earlier Duckett Jones while speaking at a New England Heart Association Cardiac Course at Boston had outlined the more frequent signs and symptoms of active rheumatic infection (Table II). As it is clear from this table, at that time the idea of major and minor criteria had not occured. Subsequently in 1938, Edward Bland (a colleague of Duckett Jones) with Duckett Jones reported 250 cases of fatal rheumatic fever. By this time (Table III) the concept of major criteria were formulated and as seen in the table so called major manifestations were mentioned. It is of interest to note that erythema marginatum was not included as a major manifestation.

Over the years, these criteria have been slightly modified mainly interchanging history of rheumatic fever or rheumatic heart disease with erythema marginatum as a major

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Table I

Manifestations in the diagnosis of rheumatic fever Duckett Jones 1944

Major	Minor
1. Carditis	FEVER
2. Arthralgia	Abdominal pain
3. Chorea	Precordial pain
4. Subcutaneous Nodules	Rashes–erythema marginatum is the most significant and might be more properly classified as major manifestation.
5. Recurrence of Rheumatic fever	Epistaxis Pulmonary findings LABORATORY findings

Table II

Frequent signs and symptoms in rheumatic fever Duckett Jones (1932)

* Joint pains of varying degrees
+ Low-grade fever
* Chorea
* Various forms of erythema multiforme
* Subcutaneous nodules
Nosebleeds
Leucocytosis
Vomiting
Unexplained weight losses
+ Prolongation of auriculoventricular conduction time by electrocardiogram
+ Increased sedimentation rate of the blood cells

* Subsequently became major and +Minor criteria.
Note absence of carditis

criteria, and today the diagnosis of rheumatic fever depends upon the presence of two major criteria or one major and two minor criteria (Table IV). Also one has to note that evidence of antecedent streptococcal infection has become a pre-requisite for the diagnosis of rheumatic fever. If this evidence is lacking and a patient fulfills the other criteria then like malaria one has to call it clinical rheumatic fever.

Rheumatic Fever in India

When the author first came to India in early February 1958, within a short period he realised that if one had to diagnose rheumatic fever according to modified Duckett Jones criteria, many cases of rheumatic fever would be missed. In 1960, the author reported his personal experience at Boston and compared them to the frequency of occurrence of major criteria in Delhi. Since then, further observations have delineated the pattern of rheumatic fever as seen in Delhi (Table V). It would thus appear that the two major criteria that are common in India are those of carditis and joint pain. So far the author has not come across a single case of erythema marginatum. Also such senior physicians as R.J. Vakil, K.L. Wig, J.C. Bannerji have not seen erythema marginatum in Indian children. Singh Paul of Delhi and some other pediatricians, on the other hand during a joint WHO-Academy Seminar on rheumatic fever and rheumatic heart disease held in February 1971, claimed to have seen a few stray cases of erythema marginatum. The total claim was that of three cases. Subcutaneous nodules and chorea occur infrequently. Later Dr. Berry of Chandigarh however believed that these were as common in Chandigarh as in Boston. Curiously enough in the last six weeks (mid July to end of August), six patients with subcutaneous nodules have been admitted to the cardiology ward.

Rheumatic Fever in Neighbouring Countries

To add the confusion, the profile of rheumatic fever shows wide variation even in the neighbouring countries (Table VI). It would appear that in Ceylon rheumatic fever is not diagnosed without objective polyarthritis, whereas in the Phillipines the diagnosis of rheumatic fever is made only on the co-existence of carditis. Curiously enough erythema marginatum also does not occur in the neighbouring countries of Ceylon and Phillipines and only rarely in Israel.

Presence of Rheumatic Activity

With this variable picture of rheumatic fever how can one diagnose the presence of rheumatic activity in young patients who already have established valvular heart disease? In this communication an attempt is being made to explore the feasibility of suspecting rheumatic activity on the presence of certain clinical and laboratory findings.

Table III

Clinical manifestations of rheumatic fever during final illness in 250 cases

	No. of Cases	Percentage
MAJOR MANIFESTATIONS (So Called)		
1. Arthritis	0	
2. Chorea	2	
3. Nodules (see text)	40	60
4. Carditis	250	100
(A) Enlargement (Dilatation)	250	100
(B) Valvulitis (Murmurs)	250	100
(C) Pericarditis (Friction rub)	47*	35

Table IV

Modified Jones criteria

Two major or one major and two minor
With evidence of recent streptococcal infection as indicated by

I. Increased ASLO or other antibodies
II. Positive throat culture
III. Evidence of recent scarlet fever

Major	Minor
	CLINICAL
1. Carditis	1. Fever
2. Polyarthritis	2. Arthralgia
3. Chorea	3. Previous RF or RHD
4. Erythema marginatum	LABORATORY
5. Subcutaneous nodules	1. Acute phase reactions increased Sedimentation rate. Positive C. R. P. Leucocytosis
	2. Prolonged PR interval

Table V

Rheumatic fever profile Boston Vs. Delhi

Manifestations	*Boston (457)*		*Delhi (223)*	
	No.	%	*No.*	%
Joint Pain	412	94	210	90
Carditis	240	53	152	68
Chorea	61	13	7	3
Erythema Marg.	48	11	0	0
Subcutane Nodules	54	12	6	3
ESR CRP	442	92	190	94
ASLO 400	376	82	156	70

Table VI

Comparing rheumatic fever profile between Boston, Delhi, Phillipines, Ceylon and Israel.

	Boston	*Delhi*	*Phillipines*	*Ceylon*	*Israel*
MAJOR CRITERIA	%	%	%	%	%
Carditis	53	68	100	50	30-80
Polyarthritis	58	22	Rare	100	Variable
Chorea	19	3	Rare	2	Rare
Erythema Marginatum	11	0	0	0	Rare
Nodules	12	3	Very Rare	Very Rare	Very Rare
MINOR CRITERIA					
Previous History	–	–	±	±	Variable
Arthralgia	90	84	Common	–	?
LABORATORY					
E S R	92	94	+	100	+
C R P	90		?	+	?
ASLO	82	80	?	–	Variable
ECG	25	16	25-33	–	?

Major Criteria :

Appearance of pericarditis or any of the existing major criteria like objective migratory polyarthritis (even polyarthralgia), appearance of subcutaneous nodules or chorea would naturally be very helpful in the diagnosis of activity in a patient with rheumatic heart disease. On the other hand, the appearance of the so called high pitched apical sea-gull murmur which at one time was thought to be due to active inflammatory process of the mitral valve (valvulitis) have been documented to have existed for months and even years. This would be rather against active valvulitis and therefore this evidence of rheumatic activity would be very questionable. Similarly the decrease in the intensity of the heart sounds in a patient with known heart disease would be also regarded as a very soft data.

Congestive Heart Failure

In earlier days, appearance of congestive heart failure in children below the age of 16 years was regarded pari-passu as rheumatic activity. A few years ago with the help of Prof. B. Ghosh Ray and Prof. L.N. Mahapatra of the Department of Microbiology an attempt was made to test this theory.

As shown in Table VII criteria for rheumatic activity were taken as four major, arthritis being replaced by joint pain and two minor criteria of fever elevated sedimentation rate or C. reactive protein as one and prolongation of PR interval to o.22 second or more as the second. These criteria were applied in 141 patients with rheumatic heart disease who were admitted in the wards of the AIIMS Hospital. As seen in Table VIII, 81 of them were below the age of 20 of whom 54 were on congestive heart failure and 27 were without. Sixty patients were above the age of 20 years, had congestive heart failure and 13 were without. It is apparent from this Table that while 29.6% of the patients with congestive heart failure below the age of 20 years had evidence of rheumatic activity, 22.2% of those who did not have congestive heart failure also showed evidence of rheumatic activity. On the other hand, while 6% of the patients in congestive heart failure above the age of 20 years showed evidence of rheumatic activity while none without congestive heart failure had rheumatic activity. Thus these findings fail to support the thesis that congestive heart failure in the young may represent rheumatic activity.*

Minor Criteria :

History of fever, evidence of inflammation like leukocytosis, increase C—reactive protein, prolongation of PR interval in ECG or other electrocardiographic abnormalities like disturbances in conduction and depression of T and ST segment, sudden appearance

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*Mathur had similar findings as shown in Table II.

Table VII

Activity manifestation

Manifestations	*With CHF (101)*		*With CHF (40)*	
	No.	%	*No.*	%
Chorea	1	1	1	3
SC NOD	2	2	0	0
Pericarditis	2	2	1	3
'P-R' 22+	4	4	0	0
Joint Pain	19	19	8	20
Fever	22	22	7	18
E S R/C R P	68	68	20	50
A S O>200	47	47	21	53

Table VIII

Incidence of rheumatic activity—cases of RHD

AGE	*Total*	*Cases Wih CHF*			*Cases Without CHF*		
		No.	*Active*	*Percentage*	*No.*	*Activity*	*Percentage*
Below 20 Years	81	54	16	29.6	27	6	22.2
Above 20 Years	80	40	47	6.4	13	0	0

Table IX

Rheumatic activity (Mathur)

AGE	*TOTAL No.*	$\overline{C}$ *CHF*			$\underline{S}$ *CHF*		
		No.	*Active*	%	*No.*	*Active*	%
Below 20 Years	105	80	46	57.5	25	16	64
Above 20 Years	128	95	18	18.9	33	16	42.1

of arrhythmia (atrial fibrillation) in an otherwise well controlled rheumatic heart disease patient should make one suspect the possibility of rheumatic activity and such patients should be evaluated on individual merits. When more than two or three of these minor criteria co-exist and the presence of other infections can be excluded, then even the minor criteria become strong presumptive evidence of rheumatic activity.

Rheumatic Activity, Endocarditis and other Infections:

When a patient with rheumatic heart disease presents with a history of fever, arthralgia, abdominal pain or epistazia embolic episodes, the treating physician has to face the problem in differentiating this illness between rheumatic activity, subacute bacterial endocarditis (SBE) and other infection. When no major criteria of rheumatic fever is present this sometimes can be a difficult and time consuming task. Nevertheless, the physician can more or less come to a correct diagnosis if few diligent observations are made. For example, marked anaemia of 7 or 8 gm% is certainly more in favour of SBE or some other infection than rheumatic activity. Similarly while the sedimentation rate will be elevated in both rheumatic activity and SBE, the leuckocyte count will be normal in the latter and elevated in rheumatic fever. Examination of 24 hours' urine on three successive days when shows more than 5 to 6 red blood cells per high power field will be again in favour of SBE or urinary tract infection and not rheumatic fever. Also rising ASLO titre is in favour of rheumatic fever. Some time back a joint study between Jewish Hospital, Long Island, New York and this Institute was conducted to evaluate the role of haptoglobin as an index of activity in rheumatic fever in two groups of population. Results showed that haptoglobin level were markedly increased in association of rheumatic activity and serial determination revealed that these levels return to normal as activity of the disease subsided. If facilities for determining haptoglobin are available, this would be another useful laboratory test to help one to differentiate between activity and SBE.

When blood cultures are positive then diagnosis of SBE becomes obvious. However, a note of caution is justified here. During the season of enteric fever, a rheumatic heart disease patient when admitted with fever of few days duration, a positive blood culture of Salmonella typhi or para typhi need not necessarily indicate Salmonella endocarditis. To label it as Salmonella SBE, other supportive evidences are essential. In the last three months, three or four such cases were admitted in the cardiology wards. It is also to be noted that in exceptional circumstances, SBE and rheumatic activity can co-exist. Over the last 15 years, two such patients were seen in the wards. Both of them had positive blood culture besides other stigmmata of SBE and such major criteria of arthritis and periocarditis. At necropsy active aschoff bodies were demonstrated in both the patients. It is also to be remembered that SBE some time can give rise to pericarditis and arthritis which are taken as dependable criteria for rheumatic activity. Nevertheless, as a general rule the combination of rheumatic activity and SBE is extremely rare. Also appearance of prolongation of P-R interval and other electrocardiographic abnormalities such as atrial fibrillation are in favour of rheumatic activity. Coming to other infections, just because a patient has rheumatic heart disease, he does not get any protection against enteric fever, urinary tract or chest infection including tuberculosis. Hence the various sources of infection should be meticulously reviewed and excluded.

CONCLUSION

The diagnosis of rheumatic activity in patients with rheumatic heart disease remains a challenge. However, if the criteria of rheumatic fever as current in India is carefully applied, the treating physician should be able to diagnose the rheumatic activity in an individual patient if he has diligently excluded SBE and other infections. The differential diagnosis of rheumatic activity, SBE and other infections are of utmost importance as successful management of the patient depends entirely on the correctness of the diagnosis.

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LIVING PATHOLOGY—A NEW HORIZON OF PATHOLOGY

BY

Dr. K. C. Basu Mallik.*

Pathology is a branch of Medicine which deals with the causes and the effects of the diseases on the body (Krumbhaar 1921). Like other sciences it has developed from the close observation of the natural phenomenon and drawing inferences from those. Prior to the Renessaince period the progress of this science was rather slow. Like its sister science Anatomy, the growth of this science was phenominal when dissection of human body for scientific study was commenced in the different Italian schools like that of Padua, Florence etc. where men like Antonio Beniveini, Geovani Morgangni and many others flourished. The science of pathology was enriched by the contributions made by these groups of workers who made close observation of the structural changes in the various diseased organs. The knowledge was mostly acquired in the autopsy room. Perhaps the greatest contribution was made by Carl Rokitansky of Vienna, who from his study of thousands of autopsies described the gross changes in different organs in various diseases.

Like scientific workers in other disciplines, pathologists do not fail to make use of various newer tools as and when these are available to further the progress of their science. When microscopes were developed it was left to a great pathologist Rudolph Virchow to employ this instrument in the study of Pathology. Microscopic Pathology developed as Pathological processes in different tissues were observed under the microscope. In recent years it has been possible to study further detailed changes in the ultrastructure due to the introduction of Electron Microscope.

If we analyse the growth of this science till a few decades back, we find that in this discipline the knowledge has been acquired mostly from the study of the dead body or dead tissues. This method suffers from a great drawback in that it fails to detect what actually happens in the living body or living tissues during the earlier phases of the disease process. For it is obvious that many changes occur after death of the individual or of the tissues to alter or to wipe out some of the effects of the disease that existed in the living tissue. This situation caused a great dissatisfaction amongst many pathologists who felt that the existing techniques were not adequate to get further information about the morbid processes taking place in a living body.

---

*Director of Health Services, West Bengal.

For, every disease has a beginning, a middle and an end. This simple fact is often forgotten and only the changes in the terminal stage are looked for, forgetting or relegating to the position of unimportance the earlier phases of the pathological process to the background. The pathologists sometimes fall a prey to this mistake. This is mostly due to the limited method of study made by pathologists. He has too frequently to depend on the autopsy or the study of the biopsy material. Naturally these specimens reveal the end process of the disease and often do not reveal the mystery of the earlier tissue changes in the disease process.

It is imperative therefore to study the sequence of events in a pathological process in living patients. This type of study alone gives one the insight into the progression of events in any pathological condition. To this study of disease process in living tissue late Sir Roy Cameron gave the name of Living Pathology.

In recent years both in the fields of Experimental Pathology as well as in Clinical medicine study of Living Pathology has yielded a rich dividend. One may cite the example of transparent chamber technique of Clark and Sandison, the study of circulation in living animal by Kinsley, Dyer & Kelly's the observation on the growth of tumour tissue by transparent chamber all of which have opened an unexplored field to the pathologist. His horizon has expanded. Nor the clinicians have lagged behind.

One of my earliest ventures in this field was when I was engaged in the study of the pathogenesis of gastric ulcer. I found that repeated injections of Pilocarpine in rat lead to gastric ulcer. These ulcers were invariably situated at the base of the mucosal folds (Fig. 1).

While searching for the cause of such location it suddenly struck me that it could be due to the muscular spasm compressing the blood vessels at that site. To test the accuracy of this hypothesis I employed the technique devised by Kinsley in his study of hepatic and splenic circulation in living animals. Observing the stomach of lightly anaesthetised rat (treated with Pilocarpine) under microscope, I noted that at the base of the mucosal folds the vessels were blanched (Fig. 2) due to compression and the mucosal ischaemia was responsible for depressed vitality and was thus exposed to the corrosive action of acid gastric juice. This study of a vital phenomenon on living animal clarified the causation of the peculiar location of the ulcer process. This point could not have been clarified unless such observation was made on living animal.

When I was engaged in the investigation of Pathogenesis of cholera, I became

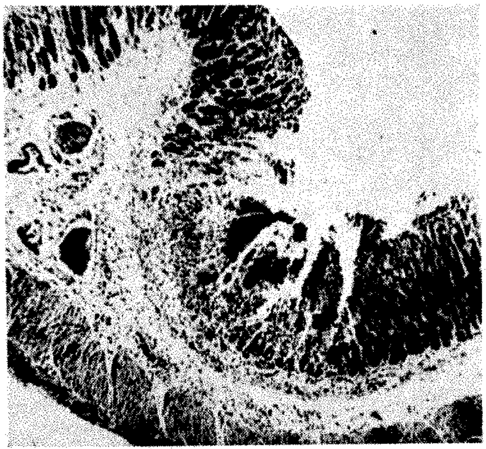

Fig. 1 Microphotograph of the section of the rat stomach showing the acute ulcer at the base of the mucosal fold. Haematoxylin & Eosin, X 60.

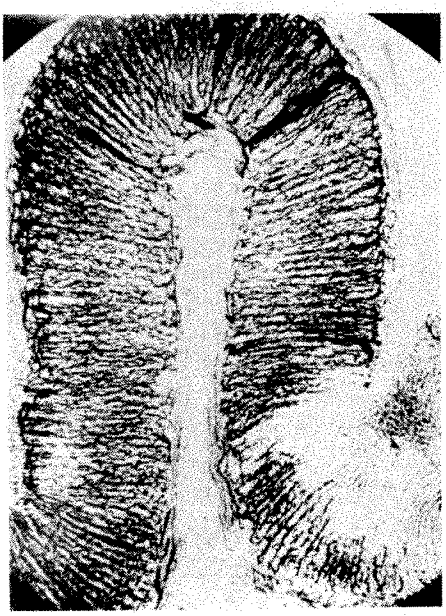

Fig. 2 Microphotograph showing the blanching of the capillaries at the base of the mucosal fold. X 60.

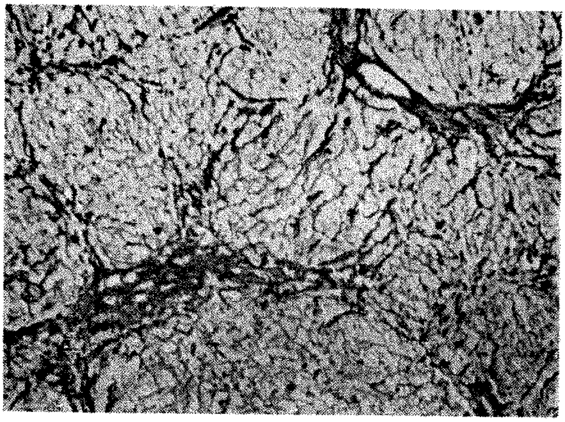

Fig. 3 Microphotograph of the section of the liver from a case of noncirrhotic portal fibrosis. Eosin & Haematoxylin. X 240.

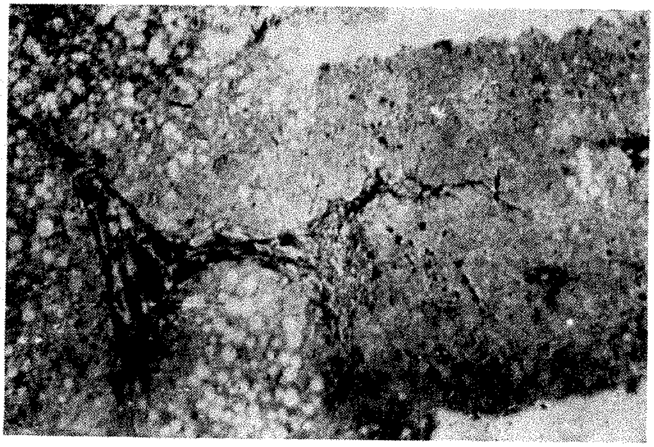

Fig. 4 Microphotograph of liver biopsy specimen from the same patient as in Fig. 3. Biopsy taken 4 years later. Haematoxylin & Eosin. X 60.

interested in the problem of lack of bile pigments in cholera stool. When we referred to the then current literature, we came across the views of Rogers (1921) who from his autopsy studies, found that there was oedema of the mucous membrane of the bile duct and he thought this is the cause of the obstruction to the flow of bile from liver into the gut. We could not aceept this view because in that case one would have expected jaundice as well amongst these patients.

It is further difficult to envisage that in a clinical condition, in which there is extreme dehydration, the mucous membrane of the bile duct on the other hand will remain oedematous. So we felt there is scope for reinvestigation of this problem.

Apart from bile pigments, other substances specially Alkaline Phosphatase is excreted along with bile. So if the hypothesis of Rogers were correct, one would have expected that there would be rise of serum Alkaline Phosphatase level in these patients. In an investigation with Prof. Mondal and Dr. Ganguly we determined the Alkaline Phosphatase level of the cholera patients and noted that though it was slightly elevated (which could be explained by the associated dehydration), it was not high enough to indicate any obstruction to its excretory pathway. Our observation was later on corroborated by other workers. Thus Carraway found high amaylase content of the stool of the cholera patients and Cole et al. noticed normal excretion of Sulphtalein in the gut of the cholera patients after intravenous administration. It is now believed that the colourless condition of the stool is partly due to dilution of the pigment and partly to the formation of the colourless pigments.

The point I would like to stress here is that we could not have arrived at this conclusion if we did not carry on our investigation on living patients. We may now pass on to another field of our investigation on cholera.

The cause for the large outpouring of fluid into gut of cholera patients remained unsolved for a long time. As a student of Pathology, it occurred to me that it could be due to an inflammatory process induced by the micro-organism. In an investigation carried out along with my colleague Dr. Ganguly, I detected that cholera stool (freshly collected by rectal catheterisation) contain an active principle which increases the capillary permeability. For the time being I was happy that perhaps we have been able to elucidate the mechanism of the fluid loss in cholera patients. But when our attention was drawn to the good work done by my predecessor Prof. Saha and late Professor A. Das, we were humbled. When they examined the fresh stool of cholera patients they found that its protein content is very low. If permeability factor were the underlying cause of the fluid loss in Cholera patients, protein content of the stool should have been high. Since the fact was otherwise, one had to look for the underlying cause elsewhere. In this connection I may draw your attention to the work of a group of workers from John Hopkins University. By their study of the fluid exchange in the gut of the Cholera patients (using radioactive isotopes) they noticed the fluid and electrolyte secretion and absorption of the cholera patients both during the acute as well as during the convalescent stage. They concluded that there is no defect in the absorptive power of the gut— the fluid loss was due to the secretion far exceeding the absorption during the acute phase of the disease. The loss of fluid is due to excess secretion of salt and water into the gut. This is a brilliant piece of work on the study of pathological process in living patients which we call living pathology.

I would now like to draw your attention to another piece of work in which I had the good fortune to be associated with Professor A.K. Basu and also my erstwhile colleagues in the Department of Pathology of Institue of Post-Graduate Medical Education and Research. While engaged in the study of the liver biopsy material of patients suffering from portal hypertention, we noted that though some sections showed typical picture of cirrhosis, the others showed either no gross change in the liver or some degree of fibrosis not amounting to cirrhosis.

When splenoportal venography was done on these patients, it was found that the former group i.e. where no gross change was detected in liver biopsy, venography suggested extrahepatic obstruction of the portal vein. But in the latter group of patients, venography was similar to that of cirrhotic cases with some minor differences. Yet repeated biopsy of liver examined over the years did not show any progression of the lesion to cirrhosis (Fig. 4).

So we were forced to conclude that these group of patients have a type of liver disease which (i) leads to portal hypertention (ii) cause fibrotic changes in the liver not amounting to cirrhosis and (iii) the lesion is stationary. This syndrome was given the name of Noncirrhotic Portal Fibrosis (NCPF). These informations could not have been acquired if we did not make serial observations on living patients. This is another venture in Living Pathology.

In this connection I would like to devote a little more time to discuss our further studies on Portal Hypertension. Portal Hypertension is caused by obstruction to the flow of blood through the portal venous system. The obstruction could be (a) suprahepatic e.g. Budd Chiari Syndrome (b) intrahepatic e.g. cirrhosis or NCPF and (c) extrahepatic e.g. cavernomatous malformation of the portal vein. Dr. Sherlock has pointed out that intrahepatic portal hypertension could be either presinusoidal or postsinusoidal. In the former case the wedged hepatic venous pressure will be normal but in the latter case it will be elevated, though splenic pressure (which is an indirect indication of portal venous pressure) will be raised in both the cases.

Disease	Plenoportal Venography	Histology	Splenic Pressure	W.H. V.P.
Extrahepatic Portal obstruction	Extrahepatic	Normal	Elevated	Normal
N.C.P.F.	Intrahepatic	Occasional Scarring	do	Moderately elevated
Cirrhosis	Intrahepatic	Cirrhosis	do	Markedly elevated.

From this study it was concluded that the obstruction to portal blood flow in NCPF must be due to post-sinusoidal block. In our light microscopic study of liver biopsy material we could find only fibrous bands usually radiating from the portal tract and splitting up the lobule up to some distance. Here I must point out one drawback of the light microscopic study of the liver sections. As mentioned earlier histology of the liver only shows a few scars in this syndrome. Now in certain conditions similar scars in the liver may be found even though the patients may not suffer from portal hypertension. Looking at the sections one cannot predict if the patient has or has not portal hypertension.

Our study by light microscope could not throw any light on the cause of the post-sinusoidal block manifested by the raised wedged hepatic venous pressure. Recently a group of workers from New Delhi have made electron miscroscopic study of liver section from the patients of NCPF. They observed that there is formation of collogen, bundles along the walls of the sinusoids. This observation if confirmed can explain the post-sinusoidal hypertension as detected by the study of hepatic venous pressure. This study clearly indicated a rewarding exercise in Living Pathology. The elucidation of a clinical syndrome, lesion identified by the pathologists and the patho-physiology unravelled by the clinicians from observation on living patients, all indicate the direction in which the future course of Pathology lies.

So long, we have been discussing the study of morbid processes in living patients. We may now turn our attention to similar study in living tissue or cell. Along with my young colleague Dr. Bagchi, I was interested in the mechanism or mode of action of certain cytotoxic drugs which are often employed as anti cancer drugs. These drugs usually act on the dividing cells and inhibit mitosis. The three drugs we selected were

Jahawerene, a crude antibiotic substance, Cyclophosphamide, a chemotherapeutic agent and Mitomycin C, a pure antibiotic. In this study we employed human leucocyte culture as the model on which the effects of these drugs were observed. The drugs were added at different stages and time intervals of culture.

It was noted that changes brought about in the human chromosomes by these cytotoxic agents were different. Jahawerene which is a crude product probably containing a number of different active substances depresses the mitosis altogether. This is probably because there is multipoint action at more than one stage in mitosis. Mitomycin C, on the other hand, causes breaks in certain large chromosomes of Group A and C, the action must be due to nonrandom action in certain specific stages of DNA synthesis. Curiously enough cyclophosphamide applied directly to the leucocyte culture had no direct effect on the chromosomes. But when serum of animals treated with cyclophosphamide or serum of patients on cyclophosphamide (Fig. 6) treatment was added, it caused pulverisation of the chromosomes. This indicates that the cyclophosphamide is metabolised and the metabolised product or products are responsible for the depressant action on the dividing cells. It would not have been possible to elucidate the mode of action of these drugs unless the study was made on living cells, the dividing human leucocytes in this case. This is another illustration in Living Pathology.

Perhaps I have given you a disjointed or patchy picture by bringing to your notice certain isolated pieces of works in which I happened to be associated. But I hope I have been able to make my point clear that on many occasions the mechanism or pathogenesis of disease processes can be understood only by the invesigations made on the living patients. In this respect workers of other disciplines have made great contributions to the progress of the science of pathology. Living Pathology is no longer the secluded preserve of Pathologists but is a living science enriched by the contribution from scientists of diverse disciplines.

Before I conclude, I would like to emphasise that the vast potentiality of Living Pathology should not eclipse the importance of autopsy room whose usefulness still exists. Every pathologist and for that matter every clinician must not neglect the autopsy room where even now many new informations may still be gathered.

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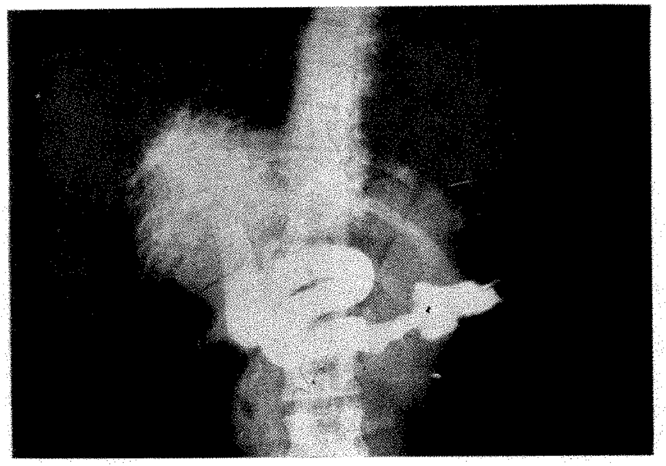

Fig. 5 Splenoportal venograph of the patient of noncirrhotic portal fibrosis. Same case whose liver biopsy section is shown in Fig. 3.

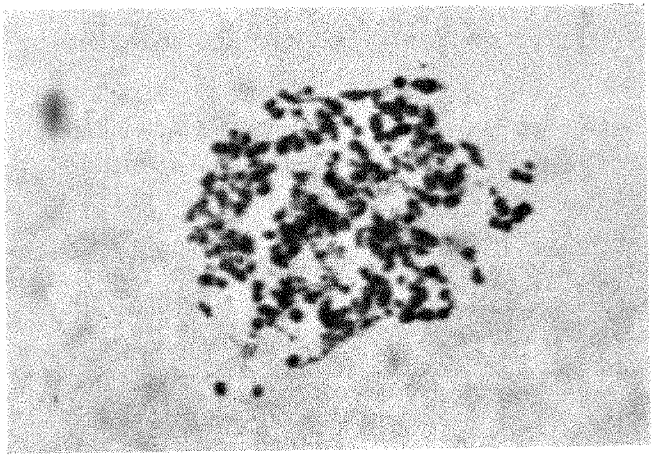

Fig. 6 Microphotograph of the chromosomes from human leucocyte culture treated with Jahawerene X 600.

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EPILEPSY : SOME PERSONAL OBSERVATIONS*

BY

Dr. K.S. Mani, B. Sc., MD, FAMS**

Epilepsy has been described from very ancient times and is an extremely common malady. Notwithstanding innumerable articles on this subject, regional variations, if any, are not without interest. This paper is based on some personal observations gathered during a prospective collaborative study on epilepsy being carried out in five centres in India with the aid of PL—480 funds.

For the purposes of this study following Williams (1964) we have defined epilepsy as "a paroxysmal, recurrent and stereotyped disturbance of movement, feeling and/or behaviour, *almost* always on a background of disturbed consciousness, such paroxysmal disturbance being primarily cerebral in origin". Only those who have had more than one attack of epilepsy in any of its varied manifestations and *without* any *clinical* evidence of progressive neurological disease at the time of first admission were chosen for this study. Those patients in whom seizures formed part of a clinical spectrum of an expanding lesion within the skullor a progressive degenerative disorder were thus excluded unless such features became obvious during follow-up.

During a two year period from 6th January 1970 to 5th January 1972 inclusive, every *fourth new* case in the out-paient department was selected (systematic random sampling), giving a total of 671 cases, of whom 40 had to be deleted for various reasons like, febrile convulsions, conversion reaction etc. Thus the total number of cases available for analysis is 631.

Males outnumbered females in the proportion of 3:2, which may reflect no more than the pattern of attendance at our out-patient. Just over the fifth of the entire group were aged below 10 years and nearly two-thirds below 20 years. 85%of the entire group were below 30 years. These figures only emphasize the well known fact that epilepsy is essentially a disorder of childhood and young adult life.

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*Based on the lecture delivered at Armed Forces Medical College, Poona on 15th December, 1973.

**Professor and Head of the Department of Neurology, All India Institute of Mental Health, Bangalore — 560027

This study was carried out with the help of a research grant HSMHA — 01—481—2 from the U. S. Government.

Being situated at the south-east corner of the state the Karnataka, Bangalore draws its patients also from neighbouring Tamil Nadu and Andhra Pradesh. Such of those who are residing outside the state of Karnataka have been excluded in the analysis of the data given in the Table 1, which indicates the rural-urban distribution of the patients with epilepsy, as also of the general population available from the 1971 census figures.

Table 1

Percentage of Rural-urban Distribution

	General Population (1971)	*Epilepsy*
Rural	76	16
Urban	24	84
Bangalore City	5	65
Other Urban	19	19

It should be emphasised that 65%of the epileptic subjects were from Bangalore city and another 19% from other urban areas, whereas the rural subjects constituted only 16%. This is quite the reverse of the rural-urban distribution in the general population and only serves to emphasize the limitations of statistics based on hospital figures.

The type of family—viz., joint or nuclear was determined and its distribution in the rural-urban group of the epileptic subjects analysed in table 2. No comparable figures for the general population are available as yet.

Table 2

Type of Family–percentage Distribution

	Joint	*Nuclear*
Bangalore City	40	60
Other Urban	43	57
Rural	51	49

Perusal of this table shows that in rural areas over 50 were from joint families and in Bangalore city they accounted for only 40%. It is also interesting to note that as time marches on, even in the traditional Indian villages, nuclear families have almost an equal distribution as against joint families, at least as far as epileptic patients are concerned. While no comparable figures are available for the general population, the impression one has is that joint families are still considerably more frequent than nuclear families in Indian villages. If this were true, the question arises as to whether epilepsy tends to cause a break-up of the joint family system even in the traditional Indian villages. This hypothetical question must remain unanswered for the present.

For the purposes of this study certain criteria were employed for the clinical diagnosis. These are indicated particularly for absence, temporal lobe epilepsy and myoclonic epilepsy, These are as follows :—

(1) Absence : Essentially a disorder of childhood and adolescence; number of untreated attacks — measured in terms of several per day. No aura or focal onset. Onset sudden, duration brief in seconds except in status absence. Usually no loss of posture but few jerky movements may be present. Incontinence of urine may be present. Terminations equally abrupt. No post-ictal phenomena.

(2) Temrporal lobe Epilepsy : Any episodic recurrent and stereotyped manifestation of psycho-sensory, psycho-motor, autonomic or psychic phenomena usually on a background of disturbed consciousness.

(3) Myoclonic Epilepsy : Paroxysmal, bilateral jerks usually without disturbance of consciousness and associated only exceptionally with major epilepsy. Since it is exceedingly difficult to distinguish between abortive major epilepsy and myoclonic epilepsy from the history, a diagnosis of myoclonic epilepsy is entertained only exceptionally in combination with major epilepsy, as for e. g., when one of the doctors had witnessed a genuine myoclonic attack.

The provisional clinical diagnosis is included in Table 3. This classification is akin to that recommended by the International League Against Epilepsy (1970).

Table 3

Provisional Clinical Diagnosis

(N–631)

Type of epilepsy	*No. of cases*	%
A. Focal :		
Motor epilepsy	7	1·10
Sensory epilepsy	10	1·58
Adversive seizures	2	0·32
Temporal lobe epilepsy	41	6·50
Other focal epilepsy	0	0·00
Indeterminate focus	1	0·16
Total	61	9·66

Contd..............................Table 3

B. Focal becoming generalised :		
Motor epilepsy becoming generalised	36	5·71
Sensory epilepsy becoming generalised	30	4·75
Adversive seizures becoming generalised	11	1·74
Temporal lobe epilepsy becoming generalised	168	26·63
Other focal epilepsy becoming generalised	0	0·00
Indeterminate focal epilepsy becoming generalised	23	3·64
Total	268	42·47
C. Generalised :		
Major epilepsy	275	43 58
Absence	4	0·64
Myoclonus	8	1·27
Others	0	0·00
Total	287	45·49
D. Combinations :		
Mojor+Absence	6	0·95
Major+Temporal lobe epilepsy	5	0·79
Major+Motor epilepsy	3	0·48
Temporal lobe epilepsy+Akinetic	1	0·16
	15	2·38
Grand Total	631	100.00

For further analysis particularly with reference to the clinical parameters, we have arbitrarily divided epilepsy into four types of attacks. These are indicated in Table 4.

Table 4
Types of Attacks
(N–659)

	TYPE	NO	%
A. Generalised at the very onset :			
(1) Major epilepsy	I	286	43
(2) Absence; myoclonic epilepsy; akinetic epilepsy	II	18	3
B. Focal epilepsy AND/OR focal epilepsy becoming generalised :			
(3) Temporal lobe epilepsy AND/OR Temporal lobe epilepsy becoming generalised	III	227	34
(4) Other focal epilepsy AND/OR Other focal epilepsy becoming generalised	IV	128	20
	Total	659	100

659 attacks in 631 subjects.

Subsequent clinical analysis is given in the form of comparative data amongst these four types of attacks.

The peak age of admission for types I and III was 15∠20 years, and for type IV attacks 20∠30 years. All cases of type II attacks were aged below 20 years. It is interesting that only 13, 16 and 22% respectively of types I, III and IV attacks were aged over 30 years.

History of parental consanguinity was obtained in 8-12% and family history of epilepsy in 17-28% of all types of attacks with no statistically significant differences amongst the four types. This information however, is meaningless unless comparable figures from a non-epileptic general population are available, a loophole which is being plugged at the moment.

There is a popular hypothesis that epilepsy is more common in the first born owing to the greater chances of foetal trauma during the first pregnancy. Data were collected not only on the pregnancy order of these subjects but also the total number of full-term live and/or still-births to their respective mothers. Information on the latter is important since theoretically most parents could have had only one child per family and epilepsy being common it has no choice but to afflict the only child. Analysis of the data available showed that in our series not only this popular hypothesis was unproven, but there was evidence to suggest that later the pregnancy order greater the chances of all types of epilepsy, such differences being statistically significant at 1 %level. Details of this aspect will be published elsewhere. (Mani, Mani, Ramesh, Kaliaperumal and Govinda Raju—1974). This contradictory finding could be due to other factors like poor obstetric care, malnourishment and childhood infections, which could conceivably be greater in later birth orders. However, this remains a matter of speculation and no more at the moment.

Table 5 indicates the number of cases with percentages with a past history of febrile convulsions and middle-ear disease in the four types of attacks :

Table 5

Past History

Past History	*I*		*II*		*III*		*IV*	
	No	%	No	%	No	%	No	%
Febrile convulsions	17	(5.94)	1	(5.56)	27	(12.27)	8	(6.25)
Middle ear and/ or mastoid disease	4	(1.40)	0		7	(3.18)	5	(3.91)

Febrile convulsions—Types I and III—Chi^2 = 5.714; df=1; $p < 0.05$
Types III and IV = N.S.

Middle ear disease—Intra—type differences N.S.

A perusal of this table shows that febrile convulsions were significantly more frequent in type III compared to type I attacks. On the other hand middle ear infection was no more frequent in type III compared to other types of attacks.

An onset of symptoms before the age of one year was very rare in types I, III and IV, whereas it was so in a third belonging to type II. An onset below the age of 20 years was slightly more frequent in types I and III, compared to type IV, but the difference was not statistically significant. No patient in type II had an age of onset over 15 years. The duration of symptoms varied from less than 3 months to over 30 years. Duration of symptoms of less than one year was distributed as 33, 33, 22 and 47% respectively in the four types of attacks. A duration of over 20 years was noticed in 3, 7 and 2% of types I, III and IV. Thus patients with temporal lobe epilepsy and/or temporal lobe epilepsy becoming generalised, tended to have a longer duration of symptoms.

Regarding the general frequency with its own limitations on the reliability of the data obtained from the Indian type of patients, subjects of type II attacks experienced several attacks a day, whereas in the other types, it was widely spread out, 60% of the patients having a frequency varying from once a week to once in three months. An increased frequency of attacks with puberty was noted in only 4—6% and with menstrual periods in only 3—10% of types I, III and IV attacks. Such a relationship was never noticed in patients with type II attacks. This increase is much more likely to be due to chance than a cause and effect relationship.

Cluster attacks (4 attacks and above in 24 hours with retention of consciousness in between) were seen in 95% of patients of type II, 27% of type I and 39% of type III and IV attacks. A history of past status epilepticus was obtained in 2-5% of all types with none in type II.

It is generally held that temporal lobe epilepsy and/or temporal lobe epilepsy becoming generalised tend to be more frequent during sleep. Our experience was quite contrary to this view. Sleep-sensitive attacks were more frequent in type I (37% as against 11% in type III and 9% in type IV attacks), this being statistically significant at 0.1% level.

Careful elucidation of various precipitating factors showed that as per history, only less than 6% of patients traced a relationship of their attacks to different phases of the lunar cycle, which is just against the widely prevalent non-medical concept. Head bath using hot water was the precipitant of a peculiar type of reflex epilepsy, the so-called Hot-water Epilepsy, in 57 of our patients (9%). This was a precipitating factor in 6, 18 and 4% of types I, III and IV attacks. This has been described for the first time in the world literature from our centre (Mani, Gopalakrishnan, Vyas and Pillai, 1968; Mani, Mani, Ramesh and Ahuja 1972; 1974). More often these attacks were temporal lobe in type. which we had confirmed by giving these patients hot water bath in our laboratory. Omission of anti-convulsants acted as the precipitating factor in only 3-5% of all types of attacks.

Amongst the various types of aura which occur in temporal lobe epilepsy, certain types were noted much more frequently than others (Table 6).

Other ictal features like epileptic cry, froth, injury (including tongue bite) and incontinence were seen in 60—80% of types I, III and IV attacks and 22% of type II. Post-ictal phenomena in the form of headache, lethargy, muscular pain, sleep etc., were invariably present in types I, III and IV attacks. (90—98%) but mostly absent in type II attacks (17%). The importance of enquiring for the presence of post-ictal phenomena cannot be emphasized too strongly in distinguishing genuine from hysterical seizures.

Table 6

Common aura in temporal lobe epilepsy (Percentages)

Vertigo	30	Epigastric Sensation	22
Dazed or Dreamy State	25	"Fear"	18
Champing of Lips	24	Automatism	17.
Visual Hallucinations	23	AuditoryHallcinations	16

Considering the criteria for selection of this material it is not surprising that abnormalities on neurological examination were noted in only 9% of types I and III and 15% of type IV and none of type II attacks. In type IV attacks, which included motor epilepsy and motor epilepsy becoming generalised minor abnormalities in the form of reflex asymmetries were more common. However, careful mental examination showed some type of abnormality or other in 10—20% of types I, III and IV and 56% of type II attacks. This high incidence of abnormalities on mental examination in type II attacks was mostly confined to myoclonic epilepsy which is known to be associated with gross brain damage.

Investigations

Barring 10 patients who did not report for EEG, all the other 621, subjects had a resting EEG with scalp electrodes. All recording were carried out on a 16 channel Galileo E.E.G. Machine using the International 10 - 20 system of electrode placement. The present data is based only on initial resting records prior to institution of drug therapy in our centre. The effect of H/V and/or P/S was noted in every case and the data included. Though we have spontaneous sleep records and records with drug induced activation with scalp electrodes and methohexitone activationwith sphenoidal clectrodes, these are not taken into consideration in this presentation. These have been described and discussed in detail elsewhere (Mani, 1973).

A resting EEG was considerd normal if the dominant activity was normal for age and alertness and there were no abnormalities in the dominant activity nor any paroxysmal discharges ; anything other than the above was considered as abnormal. It is an often repeated statement that EEG is abnormal in about 85% of patients with epilepsy and that it is an extremely important and necessary diagnostic tool. But seldom have there been any serious attempts to find out what type of abnormality one is talking of, the one notable exception being a recent paper by Ajmone Marsan (1970). When a physician wants radiological confirmation of, say mitral stenosis, he looks for certain *types* of abnormalities and not just whether the X-ray chest is normal or abnormal. Similarly, in this study, following Gloor (1965), we have defined the same criteria for abnormalities in the inter-ictal EEG which have the maximum correlation with epilepsy and are the least non-specific. Well defind spikes, sharp waves, spike and waves-generalised, unilateral or focal have alone been included as the sole criteria for a positive seizure discharge. The data on a single resting EEG in our series is indicated in Table 7.

Table 7

Single resting EEG in epilepsy

	I	II	III	IV
Abnormal	72	93	75	63
Normal	28	7	25	37
Positive Seizure Discharges :				
Present	32	73	30	13
Absent	68	27	70	87

In 63—75% of types I, III and IV attacks, EEG showed all types of abnormalities, this figure going up to 93% in type II attacks. However, when we consider the occurrence of clear cut paroxysmal definite seizure discharges, these are seen in only 32% of type I and 30% of type III attacks, the figure going up to 73% in type II and coming dowe to as low as 13% in type IV attacks. A study of possible factors associated with definite seizure discharges in interictal EEG in epilepsy (Mani, 1973), has shown that there are probably several clinical and recording parameters which influence the outcome of record. We have, for example, shown that greater the total number of attacks prior to recording greater the chances of a positive record. When patients have only 2 or 3 seizures and come for advice, as often happens in Armed Forces Medical practice, the chances of an inter-ictal EEG showing a positive record are much lower compared to those who have a much greater number of attacks. Thus the EEG is not that helpful when it is needed most. We have also shown that earlier the recording after a recent

attack, greater the chances of positive record. Hence if a patient is brought with 2 or 3 seizures, and it is desired to obtain EEG confirmation, it would be better to do the recording within 24 hours of the recent attack.

Routine radiology of the skull and chest were invariably non-contributory, being normal in over 98% of cases. We have been carrying out soft tissue radiography of the limbs in order to pick up cases of cysticercosis; the details are being published elsewhere (Mani, Ramesh, Ahuja and Mani, 1974). We found radiological evidence of cigar shaped calcification, often single in 1.4% in the soft tissues of the limbs of all patients. Biopsy of the nodules in two instances have confirmed them to be calcified cysticercus cellulosae.

Carotid angiograms on the appropriate side were carried out in 148 out of 631 subjects (23%). The findings are indicated in table 8.

Table 8

Carotid Angiograms

	Total :	*144/631*	*(23%)*		
Normal	102	(71)	Atrophy	13	(9)
Arterial occlusion	15	(10)	Fxpanding Lesion	5	(3)
		Others	9 (7)		

Arterial occlusion more common in types IV and III.

Lumbar pneumoencephalograms were carried out in 48 out of 631 patients (8%) and the findings are indicated in Table 9.

Table 9

	Total :	*48/631*	*(8.0%)*			
Normal	15	(31)		Cysticercosis	6	(13)
Atrophy	20	(42)		Others	7	(14)
Atrophy		5	2	10	3	— Selection
Cysticercosis		4	0	1	1	

Table 10 gives data on the presumed aetiological factors in these 631 subjects.

Table 10

Presumed Aetiology

(N—631)

	Not known	*540*	*(86)*		
NYD atrophy	24	(4)	Birth injury	12	(2)
Post-natal Head injury	14	(2)	Tuberculoma	2	(<1)
Cysticercosis	14	(2)	Astrocytoma	2	(<1)
Cerebrovascular Insufficiency	12	(2)	Others	11	(2)

It is worth emphasising that there were only two instances each of tuberculoma and astrocytoma, in the entire series all but one of them developing only during follow-up. Head injury was considerd significant only when there was a clear cut history of associated loss of consciousness and/or post-traumatic amnesia and/or fracture of the skull. Similarly birth injury was deemed to be significant only if there was a clear cut history of neo-natal hypoxia. By and large birth injury and post natal head injury were more often associated with type I, atrophy with type III and cysticercosis and small vessel occlusions with type IV attacks, but the figures are too small to draw any final conclusion.

SUMMARY

(1) In this prospective study on epilepsy over a period of 2 years involving 631 subjects, 65% were residents of Bangalore city.

(2) Major epilepsy accounted for 46% of all types of attacks, while temporal lobe epilepsy and/or temporal lobe epilepsy becoming generalised came next with 34%. Motor and/or motor epilepsy becoming generalised accounted for only 7%. Absence and myoclonus were very rare.

(3) There was a significant direct ralationship between pregnancy order and epilepsy.

(4) There was a significantly higher incidence of past febrile convulsions in patients with temporal lobe epilepsy — restricted or becoming generalised, than in other types.

(5) Sleep sensitive attacks were significantly more frequent in major epilepsy than in other types.

(6) Hot water epilepsy, which accounted for 9% of all our cases, was more often temporal lobe in type.

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(7) Unequivocal EEG confirmation of epilepsy from a single inter-ictal resting record was seen just in a third of patients with major epilepsy and temporal lobe epilepsy restricted or becoming generalised, under three-fourths of patients with absence and myoclonus and in about one-eighth of patients with other focal epilepsies—restricted or becoming generalised. Thus the diagnostic value of a single resting EEG in epilepsy is limited.

(8) Routine radiography of the chest, skull and soft tissue were uniformly unrewarding.

(9) Contrast radiography unearthed few instances of atrophy, arterial occlusions, cysticercosis and an occasional expanding lesion, the last mainly during follow-up.

(10) The presumed aetiology was known only in 14% of all cases, detailed investigations notwithstanding.

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A STUDY OF CHRONIC PERSISTENT JAUNDICE

BY

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Persistent jaundice which is neither haemolytic nor due to surgical obstruction though uncommon is not rare in clinical practice. Several liver diseases, namely chronic persistent hepatitis (Mallory 1947, Smetana 1957, Galleghar 1962, Sherlock 1964, 1966, Popper 1966, 1971), chronic active hepatitis (Bearn et. al. 1954, Machay et. al. 1966, Willock 1961, Sherlock 1966, Popper 1966), cholestatic hepatitis (Schaffner and Popper 1969), cholangiolitic hepatits (Mistlis 1965, 1965a), and drug hepatitis (Glober 1963, Gregory et. al. 1967, Walker 1966) can give rise to this type of presentation.

Differentiation between several of the above liver diseases presenting as chronic jaundice which is biochemically of obstructive type with predominantly conjugated hyperbilirubinaemia and raised alkaline phosphatase is often difficult. The prevalence of chronic medical jaundice with its possible aetiopathogenesis amongst patients in India has not been reported. In a hospital from Lucknow recently Mehrotra (1971) reported 333 cases of chronic liver disease which included 5 cases of chronic persistent hapatitis and 4 cases of chronic active hepatitis. The present study was planned to find out the clinical presentation, liver function tests, immunological status and histopathology of liver in patients presenting with persistent jaundice of more than 6 weeks duration.

MATERIAL AND METHOD

Selection of cases

The main criteria for selection of cases was presentation of jaundice which was either stationary or progressive for 6 weeks period. The cases of surgical obstruction of the biliary system were excluded from this study.

Method of stduy

Complete work up of the cases was done and following investigations were carried out. Serum bilirubin was estimated by the method of King and Wooton (1956). Total serum proteins and albumin were determined by Biuret Method (King and Wooton

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Table 1

Clinical Features in 11 Cases of Chronic Persistent Jaundice

Sl No.	Name	Age	Sex	Onset	Duration of Jaundice (Years)	Drug	Injection	Blood transfusion	Alcohol	Liver size (cm)	Spleen (cm)	Ascites
1	R. D.	27	F	Like I. H.	1	No	No	No	No	2	8	+
2	S. R.	31	F	Gradual with diarrhoea and weakness	7/12	No	No	No	No	2	9	+
3	K. D.	40	F	Gradual with low grade fever.	6/12	Endoxan 5 months	Yes	3 units 4 months back	No	1	–	No
4	M. D.	31	F	Like I. H.	6/12	Unani medicines	No	No	No	3	2	No
5	A. G.	24	M	Gradual	4/12	No	No	No	++	6	2	+
6	S. B.	13	F	Fever, Joint pains	5/12	No	No	No	3	5	5	No
7	A. M.	25	M	Like I. H.	5/12	No	No	No	++	3	–	No
8	Dix	53	M	Like I. H.	8/12	Rastinon	No	No	–	5	–	+
9	B. K.	35	F	Sudden with fever.	5/12	Largactil Meprobromate	No	No	No	3	–	No
10	S. S.	53	M	Like I. H.	3/12	No	Vit. B Comp.	8 units 7 weeks back.	No	3	4	No
11	V. V.	23	M	Like I. H.	3/12	No	No	No	No	1	5	+

I. H.—Infectious hepatitis.

1956). SGOT and SGPT estimations were carried out according to the method of Reitman and Frankel (1957) and alkaline phosphatase was estimated by the method of Kind and King (1954). Immunological parameters were studied by radioactive single radiation diffusion technique of Mancini (1965). Au/SH antigen and antibody were tested by the method of Prince (1968).

RESULTS

Clinical features are shown in Tables 1 and 2. There were 5 males and 6 females. Mean age for males was 37.6 years and for females 28 years. Although criteria for selection was jaundice of more than 6 weeks duration, persistent or recurrent jaundice was present for more than 3 months in all. The mean duration of jaundice was 11 months with a range of 4 months to 3 years.

Table 2

Major Clinical Features In 11 Cases of Chronic Persistent Jaundice

Symptoms	Cases	Percentage
Jaundice	11	100
Fever	9	81.8
Weight loss	8	72.7
Anorexia and nausea	7	63.2
Loose motions	6	54.5
SIGNS		
Hepatomegaly	11	100
Splenomegaly	7	63.6
Pallor	8	72.7
Ascites	5	45.4
Muscle wasting	4	36.3

The onset of illness was acute in 5, and gradual in 6 cases. Joint pains were observed in 1 patient. Haematemesis was present in 2 cases.

Table 3

Results of Liver Function Test in Chronic Persistent Jaundice

Case No.	Total bili-rubin (mg%)	Direct (mg%)	Indirect (mg%)	Total protein (mg%)	Albumin (mg%)	Globulin	SGPT millimol/ml/hour/40°C	SGOT	Alkaline phos-phatase (KA Units)
1	8.5	6.0	2.5	6.2	1.9	4.3	4.9	2.9	7.3
2	4.9	3.7	1.2	7.4	2.0	5.4	4.2	3.3	21.2
3	4.7	3.6	1.1	12.8	2.2	10.6	H	5.6	7.7
4	3.9	3.0	0.9	6.2	2.4	3.8	5.0	4.6	10.0
5	3.5	3.0	0.5	5.8	2.8	2.2	5.1	5.2	24.1
6	4.2	3.2	0.1	6.0	2.4	8.6	4.0	H	32.4
7	31.7	17.2	14.5	7.8	3.7	4.1	H	H	19.6
8	14.5	10	4.5	5.4	2.2	3.2	5.0	7.0	27.0
9	6.6	5	1.6	5.4	2.2	3.2	6.8	8.0	47.0
10	10.7	7.1	3.6	5.6	2.4	3.2	H	H	9.2
11	10.8	6.6	4.2	6.4	2.4	4.0	4.0	H	20.3

Table 4

Immunological Studies in 11 Cases of Chronic Persistent Jaundice

Sl. No.	Rheumatoid factor	TRC	"C" Reactive protein	IgG (%)	IgM (%)	IgA (%)	Serum complement level (%)	Australia antigen
1	–	–	–	158.4	116	147	52	–
2	–	–	–	55.0	200	133	53	–
3	–	–	–	308.0	275	40.6	120	–
4	–	–	–	88.0	275	18.2	60	–
5	–	–	–	110.0	275	68.6	130	–
6	–	–	–	–	71.5	51.8	–	–
7	–	–	–	–	63.8	28.7	–	–
8	–	–	–	77.0	55.0	410.6	130	–
9	–	–	–	66.0	110.0	12	140	–
10	–	–	–	–	–	–	–	–
11	–	–	–	407.0	192.5	350	150	–

Size of the liver varied from 1 cm to 6 cm with a mean of 3 cm. Spleen was palpable in 7 cases with a mean of 5 cm. Ascites was present in five cases. It was mild in 2 and moderate in 3 cases. Palmar erythema, loss of hair, finger clubbing, gynaecomastia and spider angioma were present in 2 cases.

Systemic manifestations like acne, rash, hirsutism, cushing facies, pleurisy-pericarditis, chronic glomerulonephritis, myocarditis, thyroiditis, diabetès., ulcerative colitis were not observed in any patient.

Liver Funntion Tests

The results of liver function tests are shown in Table 3.

Serum bilirubin was between 3.5 and 31.7 mg.%. Total proteins were normal, but in 10 patients globulins were definitely raised. A:G ratio was 2.4 : 4.3. SGOT and SGPT were raised in all the patients. Alkaline phosphatase was raised in 7 cases.

Immunological Studies

Rheumatoid factor was negative in all. "C" reactive proteins were positive in 2 and negative in 8 cases. IgG levels were raised in 3. IgM was raised in 5 and IgA values were raised in only 1 case. Serum complement levels were reduced in 3 cases. Immunological studies are shown in Table 4.

HISTOLOGY

In one case (No.5), the biopsy showed a moderate fatty infiltration, occasional fat cysts and focal areas of necrosis with infiltration by moderate number of neutrophil and few mononuclear cells. Cholestasis was prominent both intracellularly as well as in bile canaliculi. There was however no hyaline seen. The histological features were suggestive of alcoholic hepatitis.

In 2 patients (case No. 4 and 6) the histological features at the time of first biopsy were those described for chronic active hepatitis. The hepatocytes showed degenerative changes with ballooning of the cytoplasm and focal necrosis accompanied by mononuclear cell infiltration. These changes were more prominent in the parenchyma adjacent to portal tracts and septa. The latter were cellular and showed fibroblastic proliferation with begining of active septum formation. The subsequent biopsies in these two patients showed progression of the fibrotic process and persistence of inflammatory and degenerative changes.

In 2 patients (Case No. 7 and 8) features of persistent hepatitis were seen. There was cellularity and widening of portal tracts with small focal areas of necrosis in the lobular parenchyma. In one of these cases (Case No. 8) the necrosis was predominantly centrizonal with pronounced cholestasis. In none of these patients there was any singificant fibrosis.

In 3 patients (case No. 1, 2 and 10) the picture was, of post necrotic cirrhosis. The nodules were mostly monolobular separated by wider cellular septa. In all these cases, balloon cells, occasional councilman bodies and significant cholestasis was present. The degenerative changes and focal necrosis were more evident at the junctional area of the septa and nodule. Plasma cells were very few in all these cases. The possibility of cirrhosis being end result of chronic active hepatitis cannot be ruled out and is suggested by the presence of focal necrosis at the junctional area of the septa and the nodule.

One case (case No. 9) had histological features of surgical obstruction. The bile stasis was almost centrizonal and parenchymal infiltration was very mild. However, the exploration and operative cholangiography excluded any obstructive pathology.

One case (Case No. 3) had histological features of viral hepatitis with markedly cellular fibrous tissue.

In the remaining one liver biopsy could not be done because of prolonged prothrombin time.

The history of drug (hepatotoxic) was present in 3 patients.

One patient who was taking endoxan for the treatment of multiple myeloma, the liver histology was consistent with the histology of viral hepatitis and not drug hepatitis.

Another patient who was taking Rastinon for the treatment of Diabetes Mellitus, histological features were of persistent hepatitis.

The patient who was on Largectil (Case No. 9) for long period had histological features of surgical obstruction though on exploration no surgical obstruction was noticed.

DISCUSSION

The present study on 11 patients of chronic persistent or progressive jaundice of more than 3 months duration revealed that one case had chronic alcoholic hepatitis as liver biopsy showed moderate fatty infiltration, occasional fat cysts and focal areas of necrosis with infiltration by moderate number of neutrophil and few mononuclear cells.

The diagnosis of chronic active hepatitis was established in 2 of the 11 cases and suggested in 3 other cases.

Chronic active hepatitis is well described in Western Literature (Mackay 1956, Willock 1961, Sherlock 1966, Popper 1966) and is characterized by clinical features of recurrent or persistent jaundice gradually progressing to cirrhosis in over half of the cases with distinct predominance in females. Autoimmuno markers are prominent. Characteistic histological features are periportal piecemeal necrosis, plasma cell or lymphocytic infiltration and fibrosis in the portal area. The inflammation spills over into adjacent parenchyma, and perihepatocellular and septal fibrosis is present (De Groote et al. 1968, Popper 1970, Popper 1971, Parenette 1970, Deniach et al. 1970). In the present study two of the 11 patients have distinct histological features of chronic active hepatitis. Clinical and biochemical features are quite suggestive though not diagnostic of the disease. Three patients who presented with histological features of cirrhosis of liver had degenerative changes and focal necrosis more evident at the junctional area of the septa and the nodule, which may indicate that cirrhosis of the liver was end stage of chronic active hepatitis. It may be noticed that duration of jaundice in these patients was one year, 7 months and 6 months and serum bilirubin levels 5.5 mg. 4.9 mg. and 10.7 mg% respectively. Jaundice may be observed in the end stage of cirrhosis of liver but generally the elevation of bilirubin is of minimal degree.

Persistent hepatitis is a histological diagnosis characterised by portal hepatitis (inflammatory exudate limited to the portal area without evidence of periportal inflammation). Clinically it is a benign disease characterised by minimal features of hepatocellular dysfunction without evidence of portal hypertension. They do not show evidence of immunological disturances in the form of immunogloblulin changes or antibodies against mitochondria, [6]smooth muscle, nuclear and ductules (Mallory 1947, Popper 1966) two of the 11 patients could be grouped under this heading.

Alcoholic hepatitis is not very common in India although well described in the Western literature. Jaundice has been described in 72% of cases of alcoholic hepatitis (Mistlis and Schiff 1963) and rarely it could be as severe as described in the case in the present series (Case No. 5). Diagnosis of alcoholic hepatitis should be established on the histological criteria and not on the history of alcohol intake. It may be mentioned that in the present series another case (Case No. 7) had history of alcohol intake but liver histology in that case did not show any features of alcoholic hepatitis and the histological diagnosis was chronic persistent hepatitis.

Hepatitis due to virus B often results in persistent jaundice (Sherlock 1964). Only one case out of 11 cases studied, histologically had the features of diagnostic of viral hepatitis (Case No. 3). This patient had received repeated blood transfusions.

One of the challenging problems for the pathologist is to differentiate between extrahepatic surgical obstructive jaundice and drug hepatitis. One patient (Case No. 9) was diagnosed on classical criteria of surgical obstruction (Popper 1971) as a case of surgical jaundice. The laparotomy however failed to reveal any surgical obstruction. This patient was taking meprobromate and Largactil frequently which is a well known hepatotoxic agent (Hollister 1957).

It has been possible to give a label primarily based on histopathology of liver to the cases of chronic jaundice in this series. Chronic active hepatitis or chronic persistent hepatitis though uncommon may not be so rare in India. The data recorded, however, fails to give any clue to the aetiology of liver damage in all but 2 patients in the present series. How far the grouping of the cases into the different entities namely chronic active hepatitis, chronic persistent hepatitis, cirrhosis liver as the end result of chronic active hepatitis is going to help the clinician for the management and investigator to find out the aetiology is difficult to suggest. Classifying the patient of chronic medical jaundice on this pattern does help to predict the prognosis.

SUMMARY

Eleven cases of chronic persistent jaundice were studied. The diagnosis of chronic active hepatitis and chronic persistent hepatitis was established each in 2 cases respectively. Three cases of cirrhosis of liver were also considered to be the end result of chronic active hepatitis. Clinical picture and immunological pattern suggested the diagnosis of chronic active hepatitis in another case who did not have liver biopsy. Alcoholic hepatitis, drug hepatitis, and virus hepatitis was diagnosed each in one case of chronic persistent jaundice.

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TECHNIQUES OF TRANSFUSION

BY

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INTRODUCTION

Transfusion of blood, its components and its products have become an important established therapeutic procedure in a day to day hospital practice throughout the world. Because of its easy operative procedure and therapeutic value, there is a tendency now to misuse this valuable therapeutic measure without considering the risks involved in the transfusion. In spite of great progress in the transfusion service, yet it is not without any risk. Therefore, it is a good maxim to give transfusion only when the values accrued to a patient from such measures outweighs the inherent risks involved.

In this paper, the techniques of transfusion will be discussed in detail. This has two aspects, one in the laboratory and the other one in the ward. Both these aspects of techniques of transfusion will be considered in detail in the paper.

TECHNIQUES OF TRANSFUSION

In the Laboratory

(a) **Donor selection**—First important step in the technique of transfusion is the collection of blood. Blood donor selection is a very important step to avoid any deleterious effect occuring in the transfused patient. As a rule, the donor should be between 18 to 60 years of age and free from all diseases. History taking is very important. One of the most important things to enquire in the history is that there should not be any past history of Jaundice at least during last one to two years.

Physical examination should invariably be carried out. The donor should be healthy and free from any disease, Hb should be 13 to 14 Gm% and BP should be within normal limits. In most blood banks, Hb estimation is carried out by copper sulphate method as this is rapid and easy to perform. In border line cases, more accurate method should be used for Hb determination before rejecting a donor.

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(b) **Anticoagulants**—Preservation of blood in storage is the next important step which was accidentally improved about 28 years before when citric acid was added to citrate-glucose solution to prevent caramalisation (Loutit et al, 1943). Several modifications of the acid-citrate-dextrose mixture introduced by the above authors are now in use throughout the world.

Di sodium Citrate	2	g
Dextrose (anhydrous)	3	g
Water	120	ml

Normally we collect 420 ml blood which is mixed thoroughly with the above mentioned ACD solution. Proportion of 2·0 g disodium citrate to 420 ml of blood is adequate to prevent clotting only if the blood is well mixed with the solution during collection. The ACD solution is hypotonic and the red cells swell to about 120 per cent of their original volume. (Mollison, 1967). As this solution contains a large volume of anticogulant solution, some authors (Loutit, 1945) advocated less amount of water to an extent of 30 ml only. In U.S.A., only 67·5 ml of water is used for making the anti-coagulant solution with the same quantity of disodium citrate 2 g and dextrose 3 gm to be mixed with 450 ml of blood (NationalAcademy of Sciences, 1965).

ACD could be damaging to red cells especially in the begining (Gibson et al, 1957) they tried CPD with the following composition :

Tri Sodium Citrate	25·8	g
Citric acid	3·2	g
Sodium di hydrogen phosphate	2·18	g
Dextrose	25·0	g
Water to	1	litre

pH of this solution was 5·63 and 70 ml of this solution is used for the collection of 500 ml of blood and after equilibration pH became 7·1. This solution was found to have a better post-transfusion survival of 80 per cent at 32 days. On the whole, from the experience gained in this Centre, CPD could be considered as good as ACD from the point of view of post transfusional survival but could not be considered as better. On the contrary, survival of platelets and white cells are better in ACD than CPD. In 1954, Gabrio and Finch recorded the reparative effect of adenosine on the biochemical storage lesion of erythrocytes. As little as 1·25u mole (0·34 mg) of adenosine per ml of red cells caused a rise of ATP after incubation at 37°C whereas 25u moles produced maximum effect.

In order to increase the survival rate of the red cells, Adenine was added in a dose of 0·5u moles/ml of ACD blood (Simon, 1962). Adenine restores the ATP content of the stored red cells by incubation at 37° C. Restoration of ATP level was accompanied by an increase in the post transfusion survival. (Gabrio et al, 1956). We have found that upto a period of 45 days, red cells could safely be stored when Adenine was added to ACD. Therefore, it has become our practice to collect Rh negative blood in Adenine-ACD mixture for a longer storage. No deleterious effect has so far been noticed in transfused patients when blood collected in Adenine-ACD mixture is given. We have never tried adding inosine to Adenine-ACD mixture. No adverse reaction has also been reported by De Verdier et al (1966) in a series of 5000 transfusions of blood to which 35 mgm of Adenine per unit has been added.

(c) **Freezing of red cells**—An ideal technique for blood preservation would be to employ cryogenic temperatures to arrest the ageing of erythrocytes. In 1953, Lovelood showed that the principal damaging action of freezing and thawing is related to excessive concentration of solutes caused by partial sequestration of water in ice. It is not possible to freeze and thaw clinically useful volume of red cells rapidly enough without psing additives to prevent irreparable membrane damage. The eryoprotective compounds employed fall in two categories.

(i) Extracellular additives like PVp, dextrans, lactose, hydroxyethyl starch.

(ii) Intra cellular additives like glycerol, alcohol and other glycols.

Huggins (1964) developed a three phase general system for the preservation of blood by freezing by adding intracellular protective additives like 8 per cent glucose and 1 per cent fructose in 8·6 M ECA solution and slow freezing method at – 85° C. Frozen blood when required is thawed at 40°C and its contents are washed by glucose-fructose solution. Processing of blood before freezing and after thawing is done with the help of Huggins Cytoglomerator. Krijnen et al (1964) used glycerol and manitol as an intracellular protective additive and frozen rapidly in liquid nitrogen at – 196°C. After thawing, first washing is carried out with 16% mannitol followed by three washing with normal saline. Both the above mentioned methods have been used extensively in some countries for a long preservation of red cells and have been found to be very useful.

(d) **Packed Red Cells (PRBC)**—Concentrated or packed red cells are the major ingredient of modern transfusion practice. 70 to 80 per cent of all transfusion should be of PRBC (Greenwalt and Perry 1969). As mojority of the undesirable properties of blood lies in plasma, it is desirable to concentrate the erythrocytes which decreases the risks of circulatory overload, reduces the amount of lactic and citric acids, K, Na and citrate ions as well as anti-A/and or anti-B antibodies.

(e) **Platelet Rich Plasma (PRP) and Platelet Concentrates (PC)**—Qualitative and quantitative platelet deficiencies may be responsible for bleeding. In many institution including ours, platelet transfusion is now being given routinely to patients whose platelet count falls below 20,000 per cumm.

Platelet may be transfused as a

(i) Fresh whole blood.
(ii) Platelet rich plasma or
(iii) Platelet concentrate prepared from platelet rich plasma.

The choice between these various preparations is determined by degree of bleeding, anaemia, hypovolaemia etc. Infused platelet should be as fresh as possible, preferably within 6 hours and certainly within 24 hours of collection of blood plasma from the donors.

PRP are easy to prepare but PC, though difficult than PRP, are used more commonly because multiple transfusions are usually necessary and large volumes of plasma may result in the overloading of the circulation.

While collecting platelets, it is important to remember that blood should be collected rapidly and mixed throughly with the anticoagulant (Perkins et al, 1958). Anticoagulant of choice for Platelet preparation is ACD and it does not really matter whether a plastic bag or glass container is used (Mollison, 1917). Most authors have prepared PRP and PC at 4°C, but recently, it has been suggested that cold may enhance aggregation (Pert et al, 1917). Recently, cell separator machine is being used in many centres for the collection of platelets from a donor.

PRP prepared from a unit of a fresh blood will yield 10^{11} platelets. When these platelets are transfused to a thrombocytopenic patient with acute leukeamia the average circulating platelet increment observed is 12,000 to 14,000 per cumm/$m^2/10^{11}$ platelets given with 40 percent still in the circulation 2 days after transfusion (Freireich, 1966). PC yields less than 50 per cent of the platelet available from PRP when prepared from equivalent amounts of blood. (Levin et al, 1965)· As intravascular survival time of platelets is relatively short frequent transfusions will be required. Hence PC is preferable to PRC.

(f) **Leucocyte transfusion**—Replacement therapy with granulocytes would appear to be logical in cases of granulocytopenia and sepsis following cytotoxic therapy in malignancy. Unfortunately, procurement of granulocytes from normal donor is rather difficult without the help of a cell separator machine. With the help of this machine, it is now possible to collect enough granulocytes from one sigle donor to transfer to a granulocytopenic case. Some centres, without the availability of this machine, have utilised chronic myeloid leukaemia cases for collection of enough gralulocytes for treatment as it is rather difficult to collect sufficient number of leucocytes from normal donors. (Schwarzenberg et al, 1967). It may be of interest to note that about 40 units of normal whole blood would be required to obtain sufficient leucocytes to achieve the same increment (1000 per cumm) resulting from the transfusion of leucocytes obtained from one unit of leukaemic blood (Greenwalt and Perry, 1969). Whether such, leucocytes are comparable to normal leucocytes in combating infection is still debatable (Torynos, 1967). The percentage of patients responding seem to correlate with number of white cells administered; doses of 1×10^{11} per m^2 or more will be effective.

(g) **Fresh Frozen Plasma**—If the plasma is separated within 2 to 6 hours of collection and frozen at − 20° to − 40°C and stored at that temperature, FactorVIII activity will be maintained for several months. It has been suggested (Rizza & Biggs, 1969). Group A male donors who have a higher Factor VIII levels (approximately 6 per cent) than female donors and also 8 per cent higher than Group 0 donors, should be bled in standard ACD solutions, plasma should be separated with 2 to 6 hours of collection and frozen rapidly and stored at-30°C to-40°C. The plasma before use should be thawed immediately at 37°C avoiding vigorous agitation and frothing. Because of large volume of plasma required, it is surely possible by means of plasma therapy to raise a patients Factor VIII level above 30 per cent of average normal. Usual practice is to give an initial transfusion of 15 ml of fresh frozen plasma per Kg body wt. over a pariod of 30 to 60 minutes. (Ratnoff, 1963). As the A.H.G. factor disappears quickly from the circulation especially in presence of active bleeding or fever, it is customary to transfuse 1·5 ml of plasma per Kg every 3 or 4 hours until the symptoms subside.

(h) **Cryoprecipitate-Factor VIII & Fibrinogen**—In 1964 Pool et al found that precipitated protein fraction in the human plasma contain considerable quantity of Factor VIII. This cryoprecipitate has proved to be of considerable value in the treatment of haemophilia (Prentice et al, 1967).

This cryoprecipitate-Factor VIII can be produced in any well equipped blood bank laboratory. In principle, the method of preparation consists of rapidly freezing the plasma and then allowing it to thaw at 4°C. Cold precipitated protein are separated by centrifugalisation. The supernatant plasma may be added back to the red cells or be used for the treatment of Christmas disease as it has a high content of Factor IX. The antiheamophilic globulin (Factor VIII), in each bag of cryoprecipitated material varies widely—from 40 to 350 units, averaging 100-120 units (Prentice et al, 1967). To provide sufficient heamostasis, administration of one bag per 6 kg body wt will be sufficient.

The great advantages of cryoprecipitate are that it is a by-product of a routine blood bank, the technique of preparation is simple, and does not require elaborate equipment and the product is easy to administer and is clinically efficient.

One can reserve the cryoprecipitate for the treatment of patients undergoing major surgery or dental extraction or who have sustained serious injury. One can still use fresh frozen plasma for the treatment of spontaneous haemorrhage into muscles and joints. In these conditions raising of antihaemophilic factor to 10 to 20 units/100 ml may be sufficient to stop the spontaneous haemorrhage (Breckenbridge & Retnoff, 1968). Cryoprecipitate Factor VIII and Fibrinogen can also be transfused in cases of hypofibrinogenemia and defibrination syndromes especially following pregnancy and post-partum haemorrhage. Other plasma preparations for transfusion like albumin, gamma globulin, immune globulin, hyper immune anti-D and prothrombin-complex are not being discussed in this paper as the production of these preparations are beyond the scope of a routine blood bank laboratory.

Grouping & Cross-Matching

ABO and Rh grouping is carried out by standard procedure (Dunsford & Bowley, 1967).

Before issue of blood to a recipient, it is crossmatched with donor's blood by the following three methods :

(a) Saline.
(b) Albumin.
(c) Indirect antiglobulin test.

Only in rare cases, we carry out, in addition, crossmatching using enzyme treated red cells.

Only in planned, routine transfusion where there is enough time, these three methods of cross matching can be carried out. In urgent cases, where there is hardly much time only saline and albumin or only saline method of cross matching is carried out. In extreme emergencies, where there is no time at all even to carry out ordinary grouping of the recipients blood, low titre safe group 0 (Preferably Rh negative) blood is issued to tide over the emergency and issue properly crossed matched blood for subsequent transfusions.

In the Wards

Having decided a transfusion is necessary, one has to consider then the type of transfusion material viz. whole blood, packed cells, plasma or its fractions etc. to be given depending on the clinical condition of the case. Further, the amount has to be assessed earlier in order to inform the blood bank to arrange for the same and the rate to be transfused. Normally, the blood requisition slip gives all these informations to the blood bank excepting the rate.

Routine Procedure

Routine usual procedure is to give the blood through a vein in the anticubital fossa or veins on the back of the hand and rarely, through internal saphenous vein on the subcutaneous surface of the tibia, one to two inches proximal and lateral to the internal malleolas. In children, and in young infants, it is often found convenient to give transfusion in the scalp vein. But, intraperitoneal transfusion in young children and infants have been found to be effective and 65 to 75 per cent of the transfused red cells are absorbed within 72 hours. Because of its simplicity and easy applicability, the intraperitoneal transfusion has nearly replaced the old practice of intra-sternal or intramedullary transfusion in infants. For repeatd transfusion in young babies and infants intraperitoneal transfusion is very suitable. On some occasions, it may be necessary to cut down a vein in an infant or baby and pass a narrow plastic tubing or a metal cannula through the vein and secured with the help of ligatures for giving transfusion. For rapid replacement of blood loss in severe injury or internal damage, during surgical operation of obstetric shock, it is necessary to replace the amount lost very rapidly to prevent a fatal outcome. In such conditions, gravity flow will not be enough and a positive pressure will have to be applied by attaching a Higginson Syringe to the air line of the bottle and pump air to raise the pressure in the bottle so that about 500 ml may be given in 1 to 2 mins. Greatest danger in such procedure is air embolism. To prevent this, one must stop pumping and screw up the clamp when the level of blood is still above the filter.

Intra Arterial Transfusion

In certain desperate situation, intra arterial transfusion also has been resorted to. A convenient artery for this purpose is the femoral. The dangers of overloading the heart, air embolism and arterial thrombosis following intra arterial transfusion prevented such heroic measure excepting rarely in the operation theatre.

Exchange Transfusion

Last technique in the ward to be discussed is the "exchange transfusion". Of all the indications for exchange transfusion, commonest is its application in new born babies suffering from haemolytic disorder due to Rh incompatibility amongst parents.

The technique of exchange transfusion in new born babies has been described in detail by Dutta (1968) and Dutta et al (1970), for the exchange transfusion.

Two types of sets are being used by the author for the exchange. The first type, simpler of the two apparatus, a two way syringe with a luer lock device was used. In this, the baby is exchanged through the umbilical vein which is cut half inch from the abdominal wall and suitable polythene catheter is introduced into the vein with the help of a mosquito artery forcep. This is possible within 48 hours of birth. Beyond this period, one may have to cut down to the vein through the abdominal wall by a transverse incision just a little above the umbilicus. When umbilical vein cannot be used, exchange transfusion is carried out through the saphenous vein. Procedure for the exchange is to use alternate withdrawal and introduction of 20 ml of blood with the help of a 20 ml syringe which has two-way tap arrangement. In this, the syring has to be disconnected every time to discard the blood withdrawn from the baby and wash the syringe with heparin saline. Whilst in the other apparatus, the syringe is connected with a 3-way tap arrangement. In this, syringe is no longer necessary to be dis-connected for discarding of blood and washing as it can easily be carried out through the 3rd tap. Though the labour in the later syringe is minimised to a great extent, there is a chance for a mistake to occur unless one is very carefull. Normally, we exchange 400-500 ml of blood in the first exchange.

In certain situation like hepatic coma, exchange transfusion has been carried out in the adult patients. Here, we normally withdraw blood from a separate vein than that used for the introduction of blood. In both the situations, our practice is the alternate withdrawal and introduction method and leave behind a certain amount of deficit to avoid overloading of the circulation through error in the calculation.

CONCLUSION

This paper describes in detail the techniques of transfusion which embraces both the laboratory and ward procedures.

The laboratory techniques begin with the selection of donors and collection of blood in various anticoagulants.

Preparation of PRBC, freezing and production of plasma components like PRB. PC and cryoprecipitate-factor VIII are all part of the laboratory techniques which also include the grouping and cross-matching procedures.

Indications of blood and its products, rate and method of giving these items through various routes in patients including exchange transfusion comprise the techniques in the ward.

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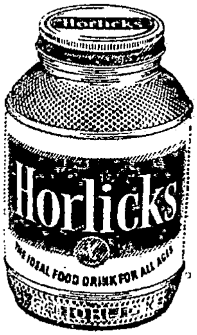

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BLOOD AND FLUID REPLACEMENT IN BATTLE CASUALTIES

BY

Colonel D. M. Gomez*

Modern concepts in the Pathophysiology of wound shock have brought about a new look in its clinical management. Attention now is focussed more on the restoration of the blood flow in the microcirculation, which, although dependant on the systemic blood pressure to some extent, can remain stagnant even in the presence of a normal blood pressure as determined by the sphygmomanometer. This is due to the opening of arterio-venous shunts in the peripheral circulation, so by-passing the capillaries, leaving the tissue cells in a state of anaerobic metabolism.

Since restoring the blood flow in the capillaries and desludging of the red cells is of primary importance for maintaining adequate tissue cell perfusion, and restoring aerobic metabolism in the cells, it would not be out of place to mention briefly the various stages of shock as reflected by changes in the microcirculation so clearly depicted in Figs 1 to 4 reproduced from Colonel RM Hardway's excellent monograph on "Clinical management of Shock". (Hardway-1968). These changes have an important bearing on fluid therapy and the response so obtained. Fig (1) shows the normal condi-

STAGES IN THE DEVELOPMENT OF HAEMORRHAGIC AND TRAUMATIC SHOCK.

(From HARDWAY RM (1968) Clinical Managemen in Shock. C.Thomas, Springfield Illinois USA)

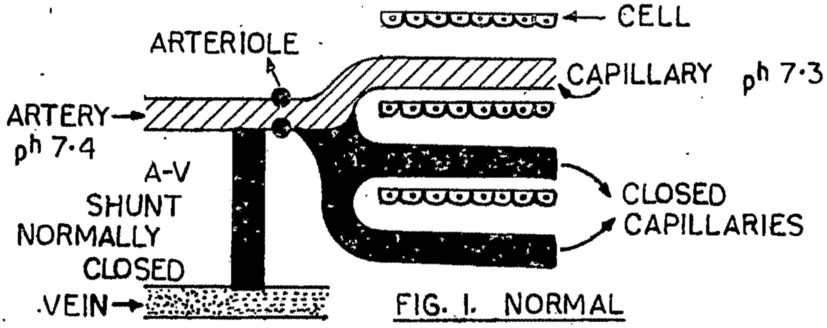

FIG. I. NORMAL

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* OC and Senior Adviser in Pathology, Southern Command, Command Pathology Laboratory (South) Pune—I

tion in the micro-circulation where it will be seen that not all the tissue capillaries are open at the same time. In fact capillary perfusion takes place in groups so that batches of cells are perfused alternately; the opening up of the pre-capillary sphincters being governed perhaps by chemical mediators liberated during cell metabolism. In Fig (2) the position in vaso-constrictive shock or compensated shock is shown, where due to release of catecholamines the arterioles are constricted and the systemic blood pressure maintained by reducing the vascular space. If corrective therapy by fluid replacement is not undertaken within an hour, the condition rapidly, progresses to the next stage of capillary dilatation, with opening up of all the capillaries and a consequent vastly expanded vascular space as shown in Fig (3). Merely replacing fluid by the amount lost is of no avail at this stage and amounts several times the blood volume has to be given. Only then, will the vascular space be adequately filled, ensuring blood flow in the micro-circulation, adequate perfusion of the cells, normal venous return, cardiac filling, and an adequate cardiac output.

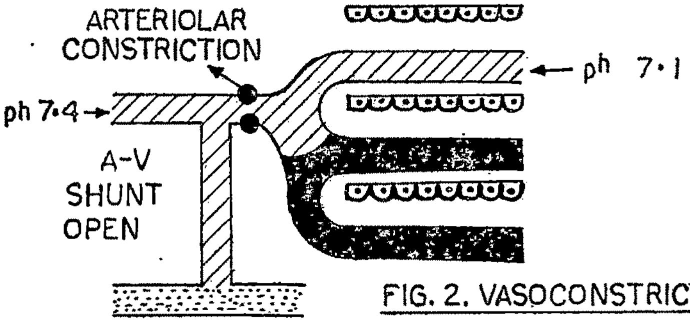

FIG. 2. VASOCONSTRICTION

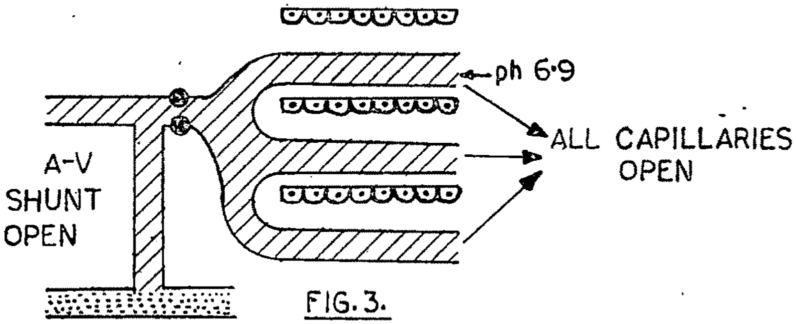

FIG. 3.
CAPILLARY DILATATION EXPANSION OF VASCULAR SPACE

If fluid replacement is not adequate, blood stasis in the capillaries takes place, with a fall in the pH and the acidotic hypercoagulable blood leads to that much dreaded complication of disseminated intra-vascular clotting, familiarly known as DIC as shown in Fig (4). The cells adjacent to these capillaries soon die and the last stage of massive cell death and organ failure is soon ushered in; the terminal or fatal event being either cardiac, renal, respiratory or liver failure. However even at the late stage represented in Fig (3), if massive amounts of fluids have been transfused and the central venous pressure is about 15 cms of water, the administration of vasodilator drugs and anti-sludging fluids like Lomodex, soon flushes out the capillaries, thus restoring the micro-circulation and systemic blood pressure. This stage can often be detected by a coagulation defect, a prolonged prothrombin time, and a decrease in the platelet count.

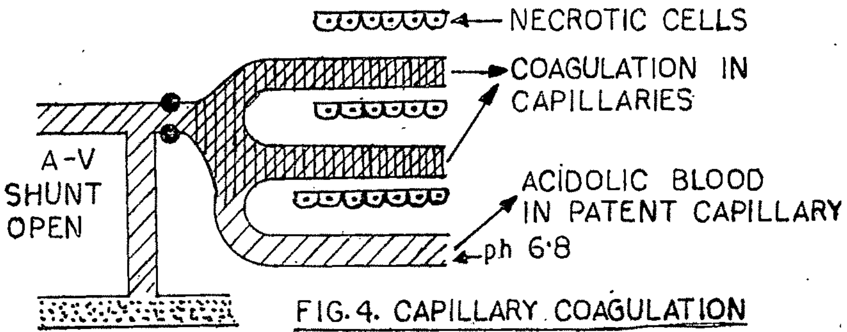
NECROTIC CELLS
COAGULATION IN CAPILLARIES
A-V SHUNT OPEN
ACIDOLIC BLOOD IN PATENT CAPILLARY
ph 6·8

FIG. 4. CAPILLARY COAGULATION (DIC)

Amount of Fluid:—With this brief outline a better appreciationof the amounts of fluid required can be made and there can be no doubt that intravenous fluid administration is the most important single factor in the treatment of oligaemic shock. In the early stages it has already been stated that it would suffice merely to replace the amount of fluid lost, and this can be assessed readily and promptly, albeit roughly by such methods as the use of the open palm in surface wounds, and of the closed fist in wounds of depth. Wallace's rules of nine roughly indicates the volume to be replaced in burn cases (Wallace-1953).

Simple parameters of adequate hydration that can be assessed clinically, are the cuff, BP, pulse rate, pulse volume, nail bed capillary blood flow, rectal/skin temperaure difference of not more than one degree centigrade (a simple index of tissue cell perfusion) and hourly urinary output of 50 to 100 ml. In Base Hospitals this can be conveniently estimated by an indwelling catheter. However, a careful watch must be kept for those cases of shock which present as intense vasoconstriction and even oliguria, due to the release of catechol amines, the body's first line of a protective mechanism. It will be fatal to assess these cases as a renal shut down and limit the fluid intake. They will rapidly progress into the later stage of expansion of the vascular space, cellular hypoxia and death. Unfortunately the only fool proof parameter to detect such a condition is the central venous pressure which can only be measured at Base Hospitals. Fluids and diureties like mannitol must be administered when there will be a lowering of the blood catecholamines as evidenced by slowing of the pulse, a warm skin. absnence of sweating and a commencing diuresis.

In the later stage of expansion of the vascular space, large amounts of fluids have to be infused monitored by serial CVP readings, blood chemistry, BP, haematocrit and urinary output. A disposable CVP manometer set can measure the central venous pressure which is the most reliable index of adequate filling up of the vascular space, and thus ensuring adequate tissue perfusion. Fluids can be introduced safely so long as the CVP does not go above 15 cm of water. In this way, a raised pulmonary artery pressure and pulmonary oedema can be avoided. If the systemic blood pressure still remains low and the CVP has reached the upper limit of 15 cms, fluids must be stopped and a vasodilator like phenoxy benzamine administered. This will be followed by a ramarkable rise in the systemic BP and an overall improvement in the general condition, due to reversion to normal aerobic metabolism in the cells, restoration of the acid base equilibrium in the tissues and blood, and a satisfactory urinary out-put.

In the modern management of aerobic shock, a case is never considered irreversible until death ensures. A useful index of progressive therapy is the blood lactate level (normal 12 to 18 mlgs) which correctly reflects the state of tissue perfusion. Unfortunately this parameter requires the facilities of a well equipped Laboratory and can only be estimated at the Base. However like all other parameters, this estimation too, has to be intelligently interpreted with regard to the clinical state of the patient for, during adqeuate fluid replacement and restoration of the flow in the microcirculation, blood lactate rises initially due to washing out of metabolites in the hitherto stagnant peripheral circulation and then declines.

Nature of fluid :—Since restoration of blood flow in the microcircluation is the primary objective, any fluid easily and immediately available can be used, ranging from a simple isotonic saline to the more complex tissue repair solution such as Ringer Lactate, and colloids such as low molecular weight Dextran (Dextran 40). Blood which presents many problems in both supply and administration is not necessary in forward areas unless the haematocrit value falls well below 30 mm. In fact it is far better to use electrolyte solutions in conjunction with low molecular weight Dextran, for there is always an element of dehydration in battle casualties and this deficiency could simultaneously be met with tissue repair solutions, a list of which is given at Fig 5. Further in the event of circulatory overload, the kidney will find it easier to excrete the excess electrolyte fluids rather than colloids. In the past, there was too much emphasis on crystalloid solutions diffusing out of the lung capillaries and causing pulmonary oedema. Now it has been shown that so long as the upper limit of the CVP is not exceeded, pulmonary oedema will not occur. Low molecular weight Dextran has already proved its efficiency by stimulating capillary flow both by its anticoagulant as well as antisludging effect, thus preventing the tendency to disseminated intravascular clotting so frequently observed in cases of shock where treatment has been delayed. It should however never be given without electrolyte solution and in no case should three bottles (1.5 Litres) be transfused at a time for fear of precipitating a dangerous bleeding diathesis. However, in the later stage of fluid replacement in field hospitals and Base hospitals where adequate facilities for blood transfusion exist, blood should be given to restore the haematocrit to the normal level.

Tissue Repair Solution

Item	Constituents	Grams Per Litre	Milli-equivalents/Litre			
			Na	K	Cl	HCO_3 or Lactate
1. Isotonic saline	Sodium chloride	9	155	–	155	–
2. Ringer lactate	Sodium lactate Sodium chloride Pot. chloride Calcium chloride	 7.5 0.4 0.4	139	5	111	28
3. Sodium lactate	Sodium lactate		167	–		167
4. Sodium bicarbonate	Sodium bicarbonate	15	174	–	–	174
5. Darrow's Repair Solution	Sodium chloride Pot. chloride Sodium lactate	4 2.7 –	121	35	104	52

Fig. 5

Acid/Base balance :—Regarding restoration of the disturbed acid base state due to anaerobic tissue cell metabolism, it has been shown from experience that by merely re-establishing the flow in the micro-circulation and ensuing adequate tissue cell perfusion, the blood pH is automatically corrected. No special alkalising fluid is therefore required. However there may be instances in the injured where a serious derangement of Acid/ Base exists, and the administration of a 1.5% solution of sodium bicarbonate is necessary, at the same time taking care not to convert an acidosis into an alkalosis which so often happens in these cases.

Organisation for Fluid replacement therapy:—To meet these theoretical councils in practice, an organisation for the supply of transfusion fluids and equipments was set up in the Rajasthan Sector in Dec 71, briefly as follows :—

(a) Blood Banks and transfusion fluids and equipment depots were set up at MH Jodhpur, MH Ahmedabad and Section Hospital Bikaner to feed the Barmer and Jaisalmer Sectors, Kutch Sector and Bikaner Sector respectively. Forward of Jodhpur, two sub depots and blood banks dependent on MH Jodhpur were set up at Barmer and Jaisalmer at the evacuation hospitals located in these towns. Blood was transported by Air/Rail in suitably insulated ice boxes with temperature holding capacity for 18 hours. This ensured that casualties were resuscitated within one hour of wounding and in any case within the critical four hours. Consequently in fact it was reported that when the cases arrived at MH Jodhpur, they were already fully resuscitated.

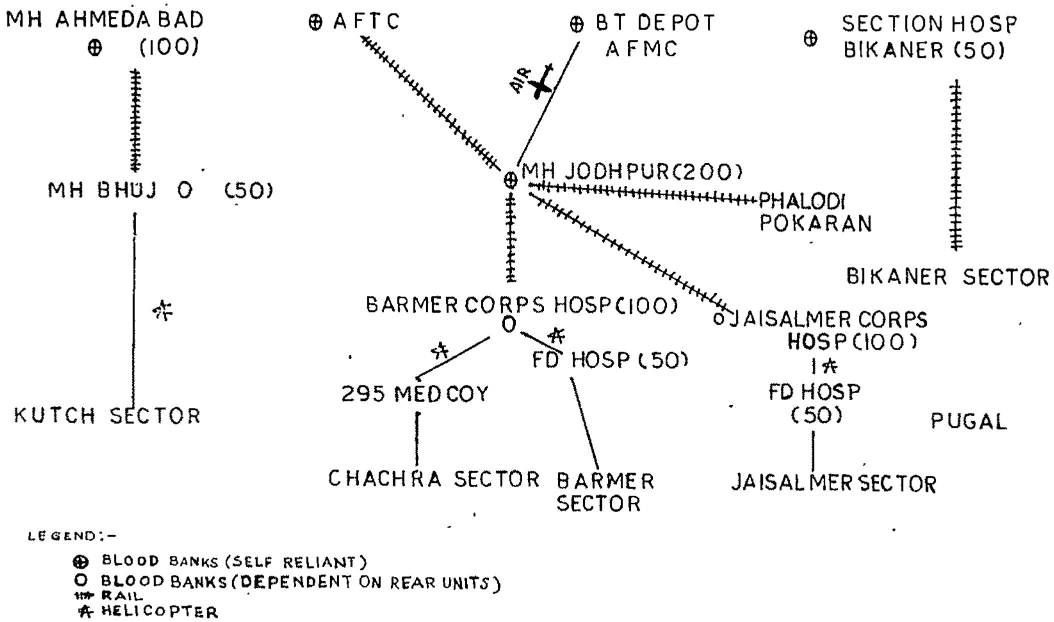

Fig. 6

(b) Provisioning of fluids for replacement therapy was based on the assessment that each battle casualty requiring resuscitation (i. e. 20% of all battle casualties) lost an average of 1000 ml blood. Fluid requirements including whole blood per case was therefore based on the formula:—total fluids required $=\frac{1}{2}$ blood loss as blood+three times blood loss as electrolyte solutions and colloids as it has been shown that the giving of blood with lactated Ringer's solution pH 8.2-8 5 is superior to blood alone in the treatment of haemorrhagic shock. (Moyer and Butcher 1967). Each case accordingly required :—

Whole blood	—1 BT bottle	(500 ml)
Ringer Lactate solution	—3 BT bottles	(1500 ml)
Glucose 5%	—1 BT bottle	(500 ml)
Lomodex	—2 BT bottles	(1000 ml)

Whole blood used was 'O' low haemolysin titre properly screened for haemolysins before labelling. This obviated blood grouping and cross matching in forward areas and was restricted to only those cases where blood loss was so severe as to bring down the haematocrit value to a figure below 30 mm.

A diagram of the supply chain of whole blood, infusion fluids and equipment adopted is shown in Fig 6.

Results achieved and lessons learnt :—Its no wonder then that the mortality rate from wound shock has been reduced considerably since the second world war. In fact it was from the experiences gained in the Korean and Vietnam Campaigns and the research conducted by Col Robert Hardway and his team in Vietnam that led to these newer concepts which in some instances even reversed clinical management based on older theories. For instance, "No longer is it just a matter of replacing blood loss, but adequate fluid administration which may be many times the total blood volume. No longer are vaso-constrictors administered for the arterioles are already constricted, but clear indications for vaso-dilators have been outlined, when the hypovolaemia is corrected. The head down position advocated so strongly in the second world war is now regarded as interfering with respiration, and the horizontal position with slightly raised extremities advised". (Hardway-1968). It may soon be that cooling the patient to lower his requirements of oxygen rather than warming may be practised as a routine. Further, reducing the volume of the vascular space by external counter pressure such as pressurised suits may be used so that what was formerly an inadequate volume, now fills the vascular space.

CONCLUSION

It will be seen from the foregoing, that in fluid replacement therapy in shock, nc one fluid can be the "be all and end all" of treatment. A judicious combination as indicated of whole colloids, tissue repair electrolyte solution and glucose, adequate in amonut and timely in administration is required to restore the blood composition to normal.

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DISEASES OF THE LYMPH NODES

(An Analysis of 1215 Cases)

BY

Lt Col B. K. Pal* and Col S. K. Chopra**

The diseases of lymph nodes are quite well known but not so their relative prevalence in different parts of our country as well as in the Armed Forces. The present communication deals with various types of lymph node lesions which have been observed in the Armed Forces personnel and their families.

MATERIALS AND METHODS

The materials consisted of biopsy and autopsy specimens of lymph nodes received in the Command Pathological Laboratory, Lucknow over a period of nearly 12 years from 1962 onwards. Metastatic malignant growths and leukaemia were excluded. Paraffin sections stained with haematoxylin and eosin were studied. Special stains like reticulin, Comori's stain for iron, Gmelin's reaction for bile pigments and Fite–Farraco stain for tubercle bacillus were used as and when indicated.

OBSERVATIONS

There were 1193 biopsy and 22 autopsy cases. These constituted 0.08 and 0.02 per cent respectively of the total biopsy and autopsy cases during the same period. An overall analysis of the lesions has been given in Table 1. There were 1136 (93.5 per cent)

Table 1

Overall Analysis of the Diseases of the Lymph Nodes

Non-neoplastic	Biopsy	Autopsy	Total	Non-neoplastic per cent
Tuberculosis	552	8	560	49.3
Nonspecific lymphadenitis	520	–	520	45.8
Tuberculoid granulomas	22	–	22	1.9
Filaria	14	–	14	1.2
Lymphogranuloma venereum	7	–	7	0.6
Others	12	1	13	1.1
Neoplastic	66	13	79	
Total number of cases	1193	22	1215	

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non-neoplastic and 79 (6·5 per cent) neoplastic lesions. Non specific lymphadenitis constituted 45.8 per cent of non-neoplastic lesions. There were 368 adult males and 49 adult females aged 17 to 60 years (average, 30 years) and 78 male and 28 female children aged 2 to 14 years (average, 8 years). The anatomical sites of biopsy are shown in Table 2. The sites were recorded in 361 cases. These were in order of frequency cervical, inguinal, mesenteric, axillary and others.

There were 560 cases of tuberculosis including 8 autopsy cases. Of these 238 were army personnels including 10 recruits. The number of their wives, sons and daughters were 212, 40 and 33 respectively. There were 37 civilians (civilians employed in the Armed Forces, Ex-army personnels, parents and personal servants of army personnels). 7 patients were officers, 6 officers wives, 5 officers' children and 3 members of the Military Nursing Services. The age of the army male patients ranged from 18 to 45 years (average, 25 years) and that of the females (excluding daughters) ranged from 16 to 45 years (average, 22 years). The youngest was a one year old male child. The sites of biopsy were recorded in 380 cases and given in Table 2. These were in order of frequency cervical (70.3 per cent), axillary (11.8 per cent), mesenteric (5 per cent), inguinal (4.2 per cent), submandibular (3.4 per cent) and others. The diagnosis was based on the presence of caseating granuloma. Tubercle bacillus could be demontsrated only in a few cases. 3 cases showed Schaumann bodies (Fig. 1).

22 cases showed tuberculoid granulomas i. e. eptheloid cells-Langhans cells granulomas without caseation, and demonstrable acid fast bacilli. 2 of these cases showed Schaumann bodies. The age ranged between 6.5 and 63 years. There were 5 females and 3 children. The sites recorded are shown in Table 2.

There were 14 cases of filariasis. 2 patients were female and 2 male children. Table 2 shows the recorded sites.

Lymphogranuloma venereum was seen in 7 cases. Only one case was female aged 16 years. The sites in all were inguinal.

Other non-neoplastic lesions observed were acute inflammation 3, suppuration 4, dermatopathic lymphadenitis 2 and bile necrosis in hepatic lymphnode in a case of obstructive jaundice due to periampullary carcinoma. In one patient aged 19 years the inguinal lymphnode showed haemosiderosis (Fig. 2) the pigments were seen mostly inside the sinus-lining cells. This patient had hepatosplenomegaly, generalised lymphadenopathy and fever. The total and differential leucocyte counts, Widal, Weil-Felix, aldehyde and Chopra tests were all negative. The biopsy of lymphnode from another site and liver biopsy were advised but not carried out. In 2 cases diagnosis of syphilis was entertained but no confirmatory serological evidence was recorded.

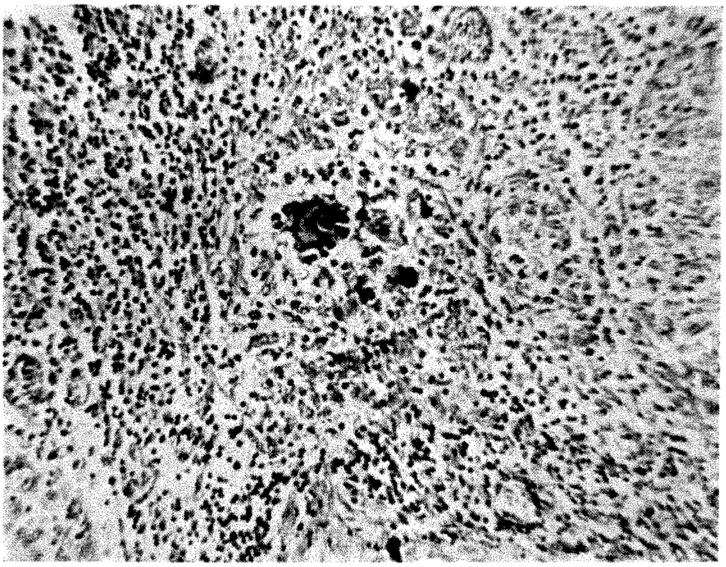

Fig. 1 Tuberculous lymphadenitis showing Schaumann's body. Haematoxylin and eosin stain. X180

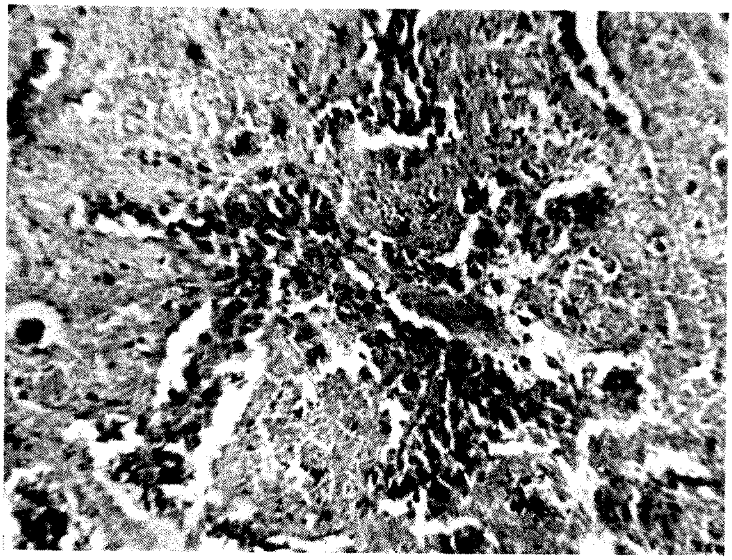

Fig. 2 Haemosiderosis of inguinal lymph node. Perl's reaction. X 180

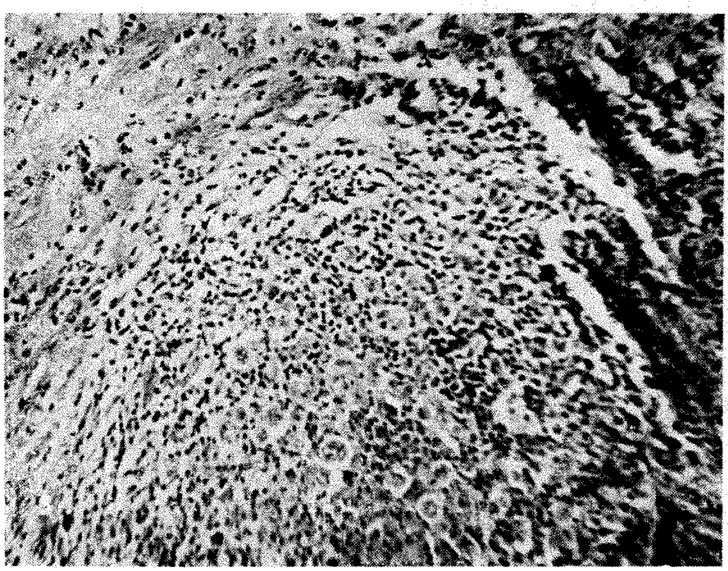

Fig. 3 Nodular sclerosis. Haematoxylin and eosin stain. X180.

Table 2

Sites of Biopsy : Nonneoplastic Lesions

Lymph nodes	Nonsp	TB	Tuberculoid	Filaria	LGV	Total
Cervical	185	267	6			458
Submandibular	10	13				23
Submental	4	7	2			13
Supraclavicular	–	5				5
Infraclavicular	–	1				1
Preauricular	3	2				5
Postauricular	3	–				3
Occipital	2	–				2
Axillary	25	45				70
Supratrochlear	5	3		3		11
Inguinal	59	16	1	4	7	87
Mesenteric	46	19	1			66
Left gastric	1	1				2
Iliac	2	1				3
Other abdominal glands	16	–	–	–	–	16
Total	361	380	10	7	7	765

Nonsp—Nonspecific lymphadenitis. TB—tuberculosis
LGV—Lymphogranuloma venereum.

There were 79 cases of malignant lymphoma including 13 autopsy cases. 46 were army personnels. There were 13 female patients. An overall analysis is shown in Table 3.

Histological classification of Rappaport (1966) for malignant lymphoma and of Lukes (1966) for Hodgkin's disease has been followed. The older nomenclature is included within parenthesis. Hodgkin's disease (all types) including 4 cases of nodular sclerosing type (Fig. 3) constituted 58.3 per cent of neoplastic lesions. The age ranged from 3 to 80 years (average 24 years). The number of other types were stem cell, 1, histiocytic 10, mixed 1, lymphocytic poorly differentiated 10, and lymphocytic well differentiated 11. One of the cases of lymphocytic poorly differentiated type was follicular type, the so-called giant follicular lymphoma which occurred in 48 years old male involving the axillary lymph glands.

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Table 3
Analysis of Lymphomas

Types	Number of Cases Biopsy	Number of Cases Autopsy	Total	Age (average)	Female	Per cent
Stem cell (R. S.)	1	–	1	46	–	1.3
Histiocytic (R.S.)	8	2	10	19-67 (38.4)	–	12.6
Mixed (R. S.)	1	–	1	28		1.3
Hodgkin's disease						
Lymphocytic predominance (PG)	9	–	9	5-34 (23.4)	–	11.4
Mixed (H. G.)	25	4	29	3-80 (25.8)	7	36.7
Lymphocytic depletion (H. S.)	2	2	4	23-35 (27.5)	–	5.1
Nodular sclerosis	4	–	4	17-25 (22.5)	2	5.1
Lymphocytic poorly differentiated (Ly. S, R. S.)	8	2	10	20-48 (32.6)	2	12.6
Lymphocytic well differentiated	8	3	11	6-53 (33.5)	2	13.9
Total	66	13	79			

R. S. — Reticulum cell sarcoma. PG—Hodgkin's paragranuloma. H.G. — Hodgkin's granuloma. H. S. — Hodgkin's sarcoma. Ly S—lymphoblastic Sarcoma.
L. S. — Lymphosarcoma.

The lymph glands involved by lymphomas, as observed from the recorded sites in biopsy cases and in autopsy cases are shown in table 4.

Table 4
The Lymph Nodes Involved by Lymphoma

Cervical	Axilla	Inguinal	Mesenteric	Illiac	Mediastinal	Abdominal lymph glands	No. of cases
+	–	–	–	–	–	–	3(B)
–	+	–	–	–	–	–	10(B)
–	–	+	–	–	–	–	2(B)
–	–	–	+	–	–	–	1(B)
–	–	–	–	+	–	–	1(B)
–	–	–	+	–	–	–	7
+	–	–	+	–	–	–	2
+	–	–	–	–	–	+	1
–	–	–	+	–	+	–	1
–	–	–	–	–	+	–	1
–	–	–	–	–	+	+	1
Total							30

(B) — Biopsy

Two cases were associated with acute myeloid leukaemia, one case of lymphocytic lymphoma well differentiated and the other, Hodgkin's disease, mixed subtype.

DISCUSSION

The data obtained in this study lead to certain important conclusions. The diseases of the lymph nodes are quite frequent in the Army. The commonest disease is the tuberculosis. Sex incidence is almost equal. The ratio of officers to non-officers is 1 to 33. This preponderant affection of the latter does not appear to be a mere reflection of their numerical preponderance but of their lower state of general health and hence of general resistance. There has not been any decline in this disease during recent years. This fact combined with the large number of cases met with by us focuses attention on the magnitude of the health problem in the Army. No group of peripheral lymph nodes has been found exempted from infection except the occipital and posterior auricular. The cervical group, as expected from the well known route of infection of tuberculosis, is the most commonly involved lymph glands. The occurrence of isolated involvement of the supratrochlear and preauricular lymph glands has been an interesting finding.

The diagnosis of tuberculosis based solely on the presence of caseation may be subject of criticism. As Millard (1966) says "it is a caseating tuberculoid granuloma awaiting a positive Ziehl-Nelsen stain or successful culture". But at present since both these methods of confirmation are regrettably unsatisfactory caseation is still regarded as almost pathognomonic of tuberculosis.

Non specific lymphadenitis is the next common lesion. This can be caused by a variety of agents whose identity is difficult to establish in any particular case. Though most of such lesions are "inocuous" in the sense that no harm will ensue, some may be early manifestation of a specfic lesion. One lymph node of a particular group may show non specific change while another from the same group or from a different site may show a specific lesion like tuberculosis or even a lymphoma. A repeat biopsy may have to be performed and a long follow up of some of these patients may be required for final assessment.

Tuberculoid granulomas form an interesting group of lymph node lesions and produce a diagnostic problem. This can be caused by many agents such as, foreign body, inanimate or animate, or an antigen liberated from disintegrated cells (Boyd, 1970). This can also be found in sarcoidosis and early tuberculosis. Sarcoidosis has to be distinguished by clinical, radiological, and biochemical findings and to be confirmed by Kveim test. A special technique of per-iodic acid-Schiff stain has been claimed to be useful (Carter, Cross and Johnson, 1969) but confirmation of this has not come out from any other worker. Tubercle bacillus is not usually demonstrable in such granulomas. We feel that in our country tuberculoid granulomas where the causative agent is not demonstrable should be regarded as tuberculous in origin and treated as such unless proved otherwise.

Though filariasis is common in our country significant enlargement of the lymph nodes which calls for their biopsy is not a common feature. In fact none of our cases was clinically suspected. The axillary and inguinal lymph glands are usually stressed as the favoured habitats of the adult worm but we have not met with a single instance of axillary glands involvement. Our study suggests that isolated enlargement of the supratrochlear glands should put the clinicians on guard.

Microscopically none of the lymph glands affected by filaria showed significant degree of varicosity, a feature commonly stressed in many text books. The enlargement of the glands was due to inflamatory reaction. Pronounced eosinophilia is a characteristic feature of such glands; this should put the pathologist on search for the parasite which may be found only after examination of multiple sections.

No special comment is warranted on lymphogranuloma venereum as in all cases the inguinal lymph glands were involved and most of the cases were clinically diagnosed.

Haemosiderosis of the inguinal lymph gland in one case has been an interesting finding. The cause remains obscure.

Classification of lymphomas has varied with different workers and hence comparison of different works on the subject is difficult. We have adopted the most recent classification as this is being increasingly followed by workers in this field and may in near future replace the others.

Though our number of cases of lymphomas is small the overall pattern agrees fairly well with those of previous workers (Table 5). Lymphomas have been found in all ages from 3 to 80 years. The most common age group, taking all types together, has been found to be 25 to 40 years. Males largely outnumber the females. Hodgkin's disease (all sub types) is the commonest type to be followed by lymphosarcoma (both types collectively).

Table 5

Incidence of Different Types of Lymphoma : Comparison With Other Publications

	Call and Mallory. (1942)*	Lumbs and Newten (1957)*	Present study (1973)
Reticulum cell sarcoma (all types)	19.4 per cent	7.7 per cent	15.2 per cent
Lymphosarcoma (Lymphoblastic and lymphocytic)	35.6	16.7	26.5
Hodgkin' paragranuloma	–	7.7	11.4
Hodgkin's granuloma	31.9	51.6	36.7
Hodgkin's sarcoma	6.05	–	5.1
Anaplastic sarcoma of lymphoid tissue	–	9.3	–
Follicular Lymphoma	6.9	6.9	0.13
Total number of cases	585	376	79

* Cited from Rappaport, 1966

Nodular sclerosis demands special attention as it offers a better prognosis than other subtypes of Hodgkin's disease. This was established as a separate entity by Lukes in 1963. The study of Cross (1968) has confirmed it but also delineated one sub-group with predominance of abnormal reticulum cells in which the prognosis is no better than in the classical type. Lukes (1963, 1966) who gathered cases from the U. S. Army found this lesions in about 39 per cent of the cases of Hodgkin's disease. But others (Hanson, 1964; Harrison, 1966) have found a much lower incidence, about 15 per cent. Our figure is about 9 per cent.

A striking contrast is the very low incidence of follicular lymphoma in our study of which we have found only one example while others (Call and Mallory, 1942; Lumb and Newten, 1957) have recorded a much higher incidence. This, at present remains unexplained.

The association of acute myeloid leukaemia in 2 cases of lymphoma has been a rare finding. This has been already reported (Pal, Chopra and Rastogi, 1973; Pal et al. 1973).

SUMMARY

An analysis of 1215 cases of the diseases of the lymph nodes from biopsy and autopsy materials has been presented. The commonest lesion is tuberculosis. This is almost equally prevalent amongst the army personnels and their families. The commonest site is the cervical lymph nodes. The next common lesion is nonspecific lymphadenitis. Tuberculoid granulomas have been observed in 1.9 per cent of cases. It is considered that these granulomas should be regarded as tuberculous unless otherwise proved. Other non-neoplastic lesions observed are filaria and LGV. Haemosiderosis of enguinal Lymph node has been seen in one case the cause of which remains obscure. Lymphomas constitute 6.5 per cent of the lymph node diseases. Hodgkin's disease is the commonest type followed by lymphosarcoma. Two cases of acute myeloid leukaemia associated with lymphoma have been observed.

ACKNOWLEDGEMENT

The authors thank collectively all Surgeons who provided the materials for this study.

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PROSTAGLANDINS-CHEMISTRY AND FUNCTIONS

(A Review Article)

Lt Col M. N. Chatterjea* and Colonel S. K. Chopra*

'Prostaglandins'—are a generic term for a family of closely related biologically active lipids. The name 'prostaglandins' was given by Von Euler in 1935. He isolated a biologically active factor from human seminal plasma. This factor was a lipid of unknown structure, which had a stimulating effect on smooth muscle and also lowered blood pressure. Subsequently, in 1936, Von Euler demonstrated substances with similar biological activity in the seminal fluid of other sepcies like monkey, sheep and goat and also in extracts of sheep seminal vesicle.

Prior to Von Euler, the existence of such a compound was first made by Kurzok and Lieb in 1930. They demonstrated that fresh human seminal fluid when applied to isolated strips of human uterus produced either contraction or relaxation.

It was not until the late 1950 that it was realised that prostaglandins is not a single substance but a mixture of several chemically related substances.

CHEMISTRY

Bergstrom carried out extensive studies to elucidate the structure of prostaglandins. He confirmed Von Euler's findings that the biological activity is due to a new group of highly active lipid soluble unsaturated hydroxy-acids.

They are all closely related derivatives of a hypothetical molecule named "prostanoic acid" (Bergstrom and Samuelsson, 1965).

Subsequently, Bergstrom and his colleagues reported that they had isolated two prostaglandins in pure crystalline form from vesicular glands of sheep. These two prostaglandins later came to be known as PGE and PGF.

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At present chemical structures of 14 naturally occurring prostaglandins are known. Chemically they are all 20 carbon fatty acids containing a "Cyclopentane ring" and chemical structure of all of them is based on the parent saturated acid "Prostanoic acid". (Fig. 1).

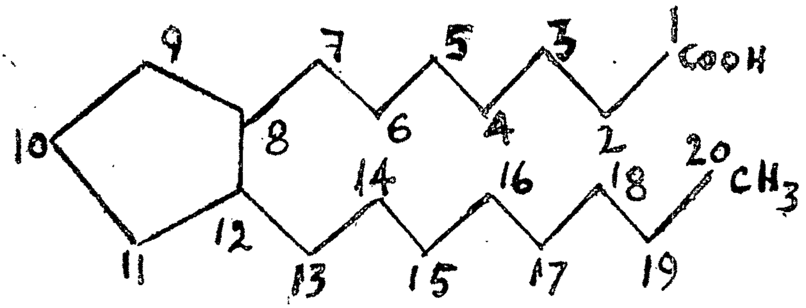

Fig. 1 Structure of prostanoic Acid.

According to their structures they can be divided into 4 main groups, viz :—

(a) PGE group — Contains PGE-1, PGE-2 and PGE-3.

(b) PGF group — Contains PGF-1α, PGF-2α, and PGF-3α

(c) PGA group — Contains PGA-1, PGA-2, 19-OH PGA, and 19-PH PG A—2.

and (d) PGB group — Contains PGB-1, PGB-2, 19-OH PGB-1 and 19-OH PGB-2.

The group are also sometimes referred to as E, F, A and B-PG. The differences in the four groups chemically are mainly in the cyclopentane ring structure. All prostaglandins have in common an OH group in 15 position and a "trans" double bond at 13 position.

The six prostaglandins of the E and F series E_1, E_2, E_3 $F_1\alpha$, $F_2\alpha$ and $F_3\alpha$ are often referred to as "Primary prostaglandins" because none is precursor of the other.

BIOSYNTHESIS

Certain unsaturated long chain fatty acids, such as linoleic acid, are essential constituents of diet (Burr and Burr, 1929). Linoleic, linolenic and arachidonic acids, as a group, cannot be synthesized in the mammalian body and must be supplied in adequate amounts in the diet in order to maintain health. These three unsaturated fatty acids are called "essential fatty acid" (EFA). However, arachidonic acid can be formed in the body from linoleic acid (Mead, 1961) by a three-stage reaction. The pathway is first by dehydrogenation of the CoA ester through γ-linolenate followed by the addition of a 2-carbon unit, probably as acetyl-CoA in the mitochondrial system for chain elongation or as malonyl-CoA in the microsomal system, (the latter appears to be more active) to give eicosatrienoate (home-γ—linolenate). The latter forms arachidonate by a further dehydrogenation (Harper, 1971).

Bergstrom, Danielsson and Samuelsson (1964) showed that dihomo-γ-linoleic acid, arachidonic acid and 5, 8, 11, 14, 17—Eicosapenta Enoic acid are transferred in high yield into PGE_1, PGE_2 and PGE_3 respectively when incubated with whole homogenates of sheep vesicular glands.

The biosynthesis is effected by an enzyme system associated with the "microsomal" fraction of the cell and a heat-stable factor present in the supernatent.

DISTRIBUTION IN TISSUES

Although prostaglandins were initially discovered in seminal plasma and vesicular glands, their distribution is not restricted to accessory genital glands and their secretions only. They have been found to occur in most of the tissues in the body. Lung tissues contain micro amounts of some prostaglandin compounds. Prostaglandins have been detected and isolated also from pancreas, kidney, brain, thymus and iris.

Recently prostaglandins have also been identified in human amniotic fluid and umbilical cord vessels (Karim, 1967). The release of a prostaglandin-like substance into the aqueous humour of rabbit eye on mechanical stimulation has also been reported.

Besides the presence of prostaglandins in various tissues, spontaneous release of prostaglandins from many sites has been demonstrated viz.

Human medullary carcinoma of thyroid,

Pheochromocytoma and

Kaposi's sarcoma.

PROSTAGLANDINS IN SEMINAL FLUID

Human semen has been shown to possess smooth muscle stimulating activity and this has been attributed mainly to the presence of prostaglandins. Prostaglandins in human semen has been shown to originate from the seminal vesicles.

Human semen has been shown to be richest in prostaglandins. It has been found to contain at least 13 different prostaglandins (exception being PGE 3γ). These have been isolated, identified and their chemical structures have been established. Together all these fractions amount to 330 µg/ml of semen.

Prostatic fluid, however, does not contain significant amounts of prostaglandin-like activity.

FATE AND METABOLISM

Distribution and fate of prostaglandins have been studied by administering "tritium"—labelled PGE, in rats. After 20 hrs of administration, 50% of the radio-activity was recovered from urine and 10% from the faeces. Prostaglandins was detected in various tissues. High concentrations of tritium were found in the kidney and the liver.

PGE_1 is completely metabolized in human body and also in animals to less polar derivatives which have been isolated from urine.

Recently an enzyme system has been demonstrated in lungs of many species which has been shown to metabolize prostaglandins.

FUNCTIONS

1. **Antihypertensive action** :—Prostaglandins have potent actions in reducing blood pressure in renal hypertensive dogs and in human hypertension (Lee, 1967).

PGE_1 is, however, ten times more potent and active in reducing blood pressure. The typical effect of PGE compounds is peripheral vasodilatation of the small arteries resulting in reduction of blood pressure. Infusion of PGE_1 causes a typical and reproducible increase in heart rate before fall in blood pressure is noticed.

2. **Action on platelets** :—Prostaglandins have been found to inhibit aggregation of human platelets.

3. **Action on gastric secretion** :—Prostaglandins reduce gastric secretion. Gastric secretion in rats and dogs following pentagastrin or histamine infusion has been found to be inhibited by prostaglandin administration (Shaw and Ramwell, 1968).

4. **Purgative action** :—Prostaglandins, when administered orally to human volunteers produced a purgative action. Williams, Karim and Sandler (1968) reported that thyroid nedullary carcinoma secretes prostaglandins into the circulation which probably accounts for the diarrhoea observed in these patients.

Prostaglandins are now suspected to be the cause of unexplained diarrhoea among breast-fed infants.

5. **Lipolytic action** :—Prostaglandins have an antilipolytic action, it inhibits the release of glycerol and free fatty acids from human subcutaneous fat incubated "in vitro" (Bergstrom and Carlsson, 1965).

The antilipolytic effects of prostaglandin E_1 may be accounted for by inhibition of the synthesis of cyclic AMP, possibly at the adenyl cyclase site (Butcher, Baird and Sutherland, 1968; Davies, 1968).

6. **Corticosteroidogensis** :—PGE_2. in twice daily doses of 200-500 μg reduced development of swelling in the ankles of adjuvant arthritic rats. A single daily injection of 400μg was also found to be active. The stimulation of corticosteroidogenesis by PGE_2 implies that the effect on adjuvant arthritis may be indirect via the adrenals.

7. **Action on C. N.-S.** :—Prostaglandins were thought to belong to the group of chemical mediators present in the C. N. System. Experimental studies on cats and chicks have shown recently that prostaglandins are released in the C. N. System from post synaptic membranes in response to stimulation by the transmitter or other substances but they are not transmitters themselves (Editorial, 1971).

Prostaglandins also act directly on the C. N. System. When injected into the cerebral ventricle of cats, PGE compounds produce stupor, and prolonged sedation bordering on unconsciousness, probably due to their action on brain hydroxy acids.

8. **Prostaglandins and birth control** :—In 1957 a smooth muscle stimulant was detected in the human menstrual fluid. It was later on confirmed that human nonpregnant uterus normally responded to seminal prostaglandins by relaxing, rather than contracting, thus possibly helping in sucking of semen. Thus prostaglandins may be concerned with either sperm transport or retention of ovum within fallopian tube until fertilization has occurred.

Excessive quantities of prostaglandins in the seminal fluid of a person or an abnormal proportion in it of PGE and PGF compounds, can lead to contraction of uterus rather than relaxation, which may be the cause of possible failure of implantation of ovum and consequently infertility. On the other hand, it is also significant that in some infertile males seminal prostaglandins level may be lower than normal (Bygdeman, Hamberg and Samuelsson, 1966).

During menstruation human uterus, actively extrudes the disintegrated endometrium by powerful co-ordinated contractions. This is attributed to menstrual prostaglandin, mainly to $PGE_2\alpha$; prostaglandins thus facilitate rythmical contractions of uterus, helping them to expel their contents. This has been utilised for birth control.

Recently, the use of PGE_2, at a rate of $0.5 \mu g/min$ for the induction of labour at or near term has been reported. The same prostaglandin in higher doses, $5 \mu g/min$ has been reported to be effective in therapeutic termination of pregnancy in the first and second trimesters.

SUMMARY

"Prostaglandins" is a new term in the field of biochemistry. The chemistry and functions of prostaglandins have been revived in brief. As seen from above, prostaglandins possess a wide range of pharmacological activities. It is active in astonishingly small doses. Physiological role in the human body is diverse and manifold. Prostaglandins are effective in therapeutic termination of unwanted pregnancy without any serious side effects. This discovery in the near future may prove to be a means to therepeutic abortion and birth control.

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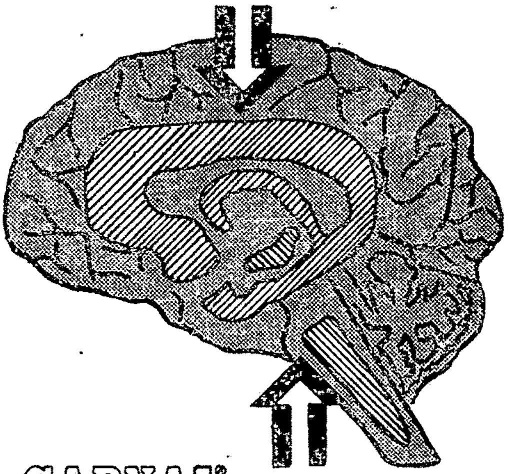

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3 BROTHERS

SOMATIC MASKS OF PSYCHIATRIC ILLNESS

BY

Capt D. S. Goel*

INTRODUCTION

The concept of psychogenicity in medicine dates back to Hippocrates, and good physicians through the ages have recognised "the importance of the patient's mind in relation to illness, and that many illnesses have no basis save in the mind. No doubt the greatest successes of physicians of past centuries were due to their ability to manage, soothe, and persuade and to apply to their patients a psychotherapy which was often intutive rather than deliberate" (Platt, 1961). It therefore appears rather strange that until a few decades back psychiatry was not only divorced from the mainstream of medicine, but was largely the preserve of quacks and charlatans. As late as half a century back, over a hundred years after Pinel and Tuke had laid the foundations of modern psychiatry by freeing mental patients from chains, there were few psychiatrists in general hospitals or medical schools, and their role was largely custodial–in big, over-crowded lunatic assylums where they, like their patients, were segregated. It was only during what Julian Huxely has called the "knowledge explosion" of the past fifty years that pioneering work in the fields of psychology, neurophysiology and psychopharmacology profoundly altered the outlook of physicians and psychiatrists alike, and by focussing attention on the hitherto poorly understood functions of the limbic system, the central reticular formation and neuroendocrine mechanisms fostered entirely new concepts such as those of psychosomatic medicine. It is sobering to realise that most modern psychiatric treatment techniques (electro-convulsive therapy, the phenothiazenes, tricyclic antidepressants; chlodiazeproxide, lithium) are scarcely three decades old, and that even now the exact aetiology of major mental illness is unclear. Perhaps the greatest impetus to advancement of psychiatric knowledge came from the two great wars, and the high incidence of psychiatric casualties (10-15% according to the History of Indian Armed Forces' Medical Services in World War II) led to phenomenal growth of military psychiatry, the work of Maxwell Jones in the U. K. and of the Meninger brothers in America are examples well known.

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The emergence of psychiatry as an important clinical speciality in the general hospital setting is paralleled by the increased awareness of somatic symptoms which are only—or mainly—psychological in origin. The skilled clinician is aware that a very considerable number of dyspepsias, aches & pains, "Heart troubles" and various vague syndromes which chronically crowd his OPD seek medication largely to fulfil their neurotic needs; some years ago an N. H. S. study in the U. K. indicated that over half of the vitamin-tonic dispensations were made mainly for their placebo effect. Needless to add, these unfortunate patients provide a rich harvest for the quack and the charlatan. The current crop of pseudo-medical articles and advertisements in the lay press ("the doctor answers", Health guide"), abound as they do with oversimplifications and overgeneralisations, have further fuelled the fire and by satisfying these neurotic demands with unneeded (though perhaps otherwise harmless) medication many myopic medical men have helped produce a large crop of iatrogenic disorders (Mayer-Gross, Slater & Roth, 1969a).

It is therefore essential that medical attention be urgently drawn to this vital, yet neglected, fields in practical terms this would involve a radical reorientation of medical education, and of the clincians who man our consultancy services. As Mayer-Gross et al. (1969 b) rightly point out, the fact that "the psychiatric aspect of medical illness can never be ignored, is not an argument for referring every patient to a psychiatrist, but for equipping with a psychiatric grounding every medical man who has to handle patients".

MATERIAL & METHOD

The present contribution attempts, albeit in a small way, to serve this end. A number of common psychiatric conditions which often masquerade under somatic masks are described and, where possible, illustrated with brief case-reports. While a detailed review of literature is outside the scope of this paper, reference has been made to the essential theoretic aspects, and the underlying psychopathologic and neurphysiological mechanisms are discussed. The classification (if it may be so called) is functional rather than formal, and is derived from the author's clinical experience in three different settings : in the dynamically oriented psychiatric unit of a major post-graduate institute, at a private psychiatric hospital catering mainly to the upper socio-educational strata (including a number of foreign patients who provided an invaluable transcultural experience), and finally as an army psychiatrist.

I Monosymptomatic conditions

(i) Hysteria
(ii) Anorexia Nervosa
(iii) Masked Depressions

II Multisymptomatic conditions

(i) Hypochondriasis
(ii) Psychophysiological Disorders
(iii) "Functional overlay"

III Unusual and "borderline" psychiatric conditions :

(i) Accident proneness
(ii) Self-inflicted Disorders
(iii) Malingering

DESCRIPTION & OBSERVATIONS

I Monosymptomatic Conditions :

These include conditions which usually present with an apparently serious or dramatic somatic symptom which at first glance appears to overshadow the wohle clinical picture. A detailed inquiry, however, usually reveals associated or underlying features which lead to the correct diagnosis.

(i) Hysteria : It has been an enduring dictum that hysteria can mimic any illness, medical or psychiatric, and indeed it does present in a bewildering variety of ways. The patient of hysterical conversion reaction has a prominant somatic symptom, usually with the history of a sudden, dramatic onset following an emotional crisis, for which no organic basis can be detected and which is rooted in psychological conflict, trauma or frustration. The symptom may involve and be mediated through voluntary as well as autonomic sensorymotor systems (Nemiah, 1971); "fits" of various types, sometimes closely simulating epilepsy are common in our country, and Abse (1959) noted their high incidence among Indian soldiers in World War II. In a recent study D'Netto (1973) has pointed out that hysterical conversion reaction is a frequent manifestation of psychiatric breakdown amongst troops in high altitude areas, and Bagadia, Shastri & Shah (1973) found that in males incidence was higher among young (16-25 years) unmarried subjects with lower education. A significant sexual element in the psychopathology of hysteria has been recognised since the time of Plato and Hippocrates, and Freud elaborated this theme in the framework psychoanalysis; Meninger (1938) introduced the concept of "functional suicide"—symbolic surrender of the function of an important organ in reparation for subconscious guilt. In our patients, however, oedipal guilt does not appear so prominently in psychopathology, and preconscious factors seem to play a more significant role.

In hysterical dissociation reaction the underlying psychological conflict leads to a molar splitting of consciousness, whereby a part of it is dissociated (or split-off, suppressed) from awareness, manifesting clinically as lacunar amnesias, paramnesias, trance and fugue states, somnambulism, highway hypnosis, multiple personality. Psychologically it may be regarded as a form of defensive escape from unbearable anxiety. The role of the reticular activating system and the diffuse thalamo-cortical projections in its neurophysiology has received increasing attention in recent years (West, 1971). This condition has aroused comparativiely little interest in our country but is, nevertheless of considerable importance as it may sometimes be difficult to distinguish from certain epileptiform phenomena, and might explain some puzzling problems. Thus highway hypnosis may be an important cause of vehicle accidents, particularly among drivers on long convoy duties, and could constitute a fruitful area of research in military psychiatry. Some of the gross stress reactions of war have a singificant dissociative element, and deserters often claim that they were wandering around in a "trance", sometimes posing a perplexing problem for the army psychiatrist.

The clinical aspects of hysteria are too well known to require repitition, but the essential criteria in its diagnosis need to be restated with precision and clarity, in order to avoid the all too frequent diagnostic pitfalls. These may be described as three major and three minor ones :

Major Criteria

(a) Absence of organic disease
(b) Recognition of the underlying Psychopathology
(c) Identification of primary and secondary gain.

Minor Criteria

(a) "La belle" indifference
(b) hysterical premorbid personality
(c) Illogicality of the clinical picture

Major Criteria

(a) Absence of Organic disease : Before diagnosing hysteria, a careful physical examination and investigation must be undertaken to exclude any organic disease; in fact this must precede every psychiatric referral (DG AFMS Medical Memorandum No. 36, 1963). This is not to say that hysteria is a diagnosis of exclusion, except to the extent that any definitive medical diagnosis implies that other diseases producing a simlar clinical picture have been excluded. A judicious balance has to be struck between investigations which are essential and those that are not, and may in fact implant more firmly the fear of physical disease in the mind of the patient.

(b) **Recognition of the underlying psychopathology** : This is a vital step without which the diagnosis of hysteria is not complete, and therapy unsatisfactory. In a majority of cases patience, sympathy and an understanding attitude would soon yield the desired information; in others specialised techniques such as narcoanalysis under Sodium Amytal (or Pentothal) and Methedrine may by required.

(c) **Identification of the primary & secondary gain** : Related to psychopathology is the primary gain (relief from anxiety caused by the emotional conflict, temporary escape from a difficult situation), and secondary gain in the form of attention sympathy, rest etc. These motivating factors are fairly transparent in most hysterics, and if the individual stands to 'gain' nothing, subconciously or otherwise, then the diagnosis of hysteria must be reviewed.

Minor Criteria

(a) **"La belle" indifference** : While the patient tries to attract attention and sympathy in various ways, he shows a curious lack of concern for his main (apparently severe) symptom. This feature (originally described in young French girls who were particularly prone to hysteria) is highly characteristic and may give the first clue to diagnosis, but it may also be seen in some cases of brain-tumour, disseminated sclerosis, arteriosclerotic brain disease, G. P. I. and schizophrenia.

(b) **Hysterical premorbid personality** : The hysterionic personality is supposed to have all the attributes of a coquettish young woman: an immature, over reacting type of personality with tendency to dramatise, attention seeking and seductive, but with a childhood type of craving for affection rather than adult sexuality. The individual has poor tolerance to stress and tries to escape from difficult situations, rather than face the problem with courage. While the personality-background of the patient is of help in evaluating the total clinical picture, it should not be forgotton that the hysterical individual can also develop serious physical illness, and that hysterical threats of suicide sometimes succeed, albeit accidentally.

(c) **Illogicality of the clinical picture** : This is the favourite criteria of medical men for making a diagnosis of hysteria; it is also the most misleading in many cases. It is true that the clinical picture of disease, specially in neurological disorders, is highly specific in several cases, and an absurd symptomatology points to a "functional" illness; but as every experienced clinician is aware, organic disease often presents in atypical and unusual forms and every patient can not be expected to fit in conveniently with the text-book picture. The consequences of this misconception are frequently tragic.

CASE REPORTS

Case No. 1 : An army jawan, in his mid-twenties, was transferred to the psychiatric centre of a command hospital as he was reported to have had a "fit", while his unit was moving to a forward area sometime before the Dec. 71 operations. History revealed that he was an "old case of neurosis" manifesting with a variety of vague neurological symptoms, and had been in low medical category for nearly two years. On examination the neurological picture was extremely confusing and variable. However, since there was no apparent motivating factor for a hysterical illness, further investigations were undertaken. The second lumbar-puncture the first having been traumatic) revealed a CSF picture characteristic of tubercular brain disease. The patient was immediately put on antitubercular therapy and steroids, but died about two months later.

Case No. 2 : A 31 year old Pnr. NCO was transferred to a base hospital with the diagnosis of "functional tremor". The patient gave the h/o occasional occipital headache and slowly progressive tremor of right hand (which had extended to the left about three months earlier) for the previous 2-3 years. O/E there was no convincing neurological deficit but, at the same time, there was no other hysterical feature either. X-ray of skull was normal but CSF examination revealed a tubercular pathology. The patient showed some improvement with antitubercular drugs and steroids, and was subsequently transferred to a neurological centre for further investigations (? tuberculoma).

Case No. 3 : A young Gurkha soldier with only a year's service was admitted to an M. H. with pain in the epigastrium aggravated on lying down. Since the medical specialist did not find any physical abnormality; he was shown to a visiting psycyiatrist who made a diagnosis of hysteria. The patient was transferred to a psychiatric centre, where two disturbing features were noted;–he had a persistantly raised ESR, and he begged to be allowed to continue in service–he had nothing to gain from a hysterical symptom. After considerable debate a barium study was undertaken, and it revealed a huge hiatus hernia. The pt. was then referred to the surgeon, who detected a large parietal abscess in the epigastric region and drained nearly 700 ml. of pus. The hernia was repaired and the individual continued in service.

Case No. 4 : A 39 year old infantry havildar with 19 year's service was admitted to the medical side with pain in abdomen, loss of appetite and feeling of ill-health. Since the clinical and investigative findings did not fit in with a definite medical diagnosis and as the patient was supposed to be making too much of his symptoms he was referred to the psychiatrist who could not find any psychological factors to account for the illness and felt that the anxiety/depression seen in the case were secondary to the organic illness. A brief (9 day) therapeutic trial with antidepressants and modified insulin having proved unsuccessful, the patient was again reviewed by the physicians who put him on antitubercular treatment. There was no improvement and a detailed barium study was undertaken; it indicated a suspicious displacement of the stomach, and lapratomy revealed a large retroperitoneal mass, subsequently proved to be malignant. The patient died a few days later.

(ii) **Anorexia Nervosa & Obesity :** Though clinical opposites, the psychopathology of these conditions is interrelated: in the former there is extreme denial of orality, which is subconsciously identified with (socially unacceptable) sexual aggression, whille the latter condition is associated with excessive oral needs. Both are (at least partly) socially determined: the anorexic girl usually starts dieting to achieve a fashionably slim figure, whereas the obese individual often overeats to (subconsciously) overcome social-emotional frustration.

The treatment is mainly psychotherapeutic with the social aspect being carefully explored, but drugs(tranquillisers, antidepressants) play an important supportive role and MIT (modified insulin therapy) is invaluable in case of anorexia nervosa. It must be realised that in clinical practice the patient may present with variable degrees of under-eating, rather that complete loss of appetite, and diagnosis should not await the development of a full blown clinical picture. The management of obesity assumes added urgency in individuals with concomitant cardiac dissease or hypertension, and is an important risk-factor in their aetiology.

Case No. 5 : The teenage daughter of an army officer was referred for psychiatric examination as she was eating very little and had spasms of uncontrollable coughing; investigation for pul. tuberculosis etc. had been non-productive. Psychiatric inquiry revealed some difficulties at school and emotional conflict arising from the adolescent surge of sexuality. She improved with narcoanalysis sessions and psycho-therapy, but developed "fits" following a fresh traumatic experience (accidentally seeing parental intercourse). She gradually made a full recovery and returned to school.

(iii) **Masked Depression :** The association of various somatic complaints with depression is well documented (Lesse, 1967; Kreitman, Sainsbury, Pearce & Costain, 1965; Robertson, 1954) since the time of Kraeplin who described hypochondriacal preoccupation in depressed patients. As every psychiatrist is aware, most depressions are marked by physical symptoms in varying degrees, and Singh (1968) found that in a series of 232 depressed patients as many as 150 (65%) had presented primarily with somatic complaints. The concept of masked depression (or depressive equivalents) has been elaborated by Lesse (1968) and Singh & Verma (1971) have described several cases in the Indian setting. The patient, often a midde-aged well educated individual with an obssessoid (conscentious, punctual, rigid, perfectionistic) personality make-up, usually presents with pain–headache (hemicrania, trigeminal type of neuralgia, migraine, facial pain), pain in chest (often with a feeling of constriction and fear of death due to heart-attack) or in abdomen, low backache– for which no organic basis can be detected; the stable premorbid, personality and lack of any motivating factor rule out hysteria. Detailed psychiatric enquiry unmasks the underlying depression, which may be of suicidal depth and which is related to a severely repressed psychic conflict or feelings of guilt, sometimes with a sexual basis. A possible diencephalic-hypothalmic mechanism has been postulated to explain the pathophysiology of this psychogenic pain.

These individuals must be recognised early from the many intractable cases of pain which frequent the medical OPD's sometimes for years, without relief. Narcoanalysis (antidepressants, tranquillisers, ECT in some cases) should be immediately instituted as there is considerable risk of suicide in these cases once the depression is unmasked–and experienced acutely as psychological pain. It must, however, be emphasised that this age group also carries a higher risk of neoplastic disease, and a brain tumour may produce depression as well as headache; hence the need for a careful neurological assessment of each patient.

Case No. 6 : The 46 year old wife of a JCO had been suffering from intractable headache for over three years (in spite of various medical treatments) till finally she was referred for psychiatric assessment. This revealed an underlying depression with involutional features, and the patient started improving with treatment (ECT under G. A. antidepressants and tranquillisers along with supportive psychotherapy).

Case No. 7 : A 28 year old sepoy was referred for a severe headache and associated vertigo for which no organic basis could be detected. Psychiatric examination revealed an underlying depression, though the patient denied it and attributed it to the result of the headache, rather than its cause. He was put on antidepressants, tranquillisers and modified insulin, and his headache was gradually relieved; as this symptom receded his depression became more evident, with silent weeping and pessimistic ideation. With further treatment the depression also improved. Psychiatric inquiry revealed that his illness had been precipitated by ingestion of cannable two months earlier (his first encounter with the stuff) and he had been afraid of its consequences.

II Multisymptomatic Conditions : Most physicians have encountered—and frequently felt fed up with—the patient whose complaint never seem to end, and involve almost every system in the body. He is the unwanted patient, a chronic attender of various clinics, taking up valuable time and overloading the already overextended investigative facilities, who is shuttled from the specialist to another till someone in exasperation makes a seemingly punitive (in the eyes of the patient at least) referral to the "mental ward". A professor of medicine in the author's undergraduate days had a simple rule of the thumb to deal with such cases: after the patient had enumerated five complaints, the learned physician would ask a leading question regarding the sixth—and an affirmative reply straightaway lead to a psychiatric referral.

(i) **Hypochondriasis :** may be defined as exaggerated concern and preoccupation with bodily functions and somatic symptoms for which no organic basis can be detected. Though his symptoms are psychogenic, the individual is convinced that he has some physical illness and resorts to a variety of treatments, surgical as well as medical; many an innocent appendix has thus been sacrificed. Physicians often find it tempting to treat these individuals with bland reassurance and placebo prescriptions, thereby implanting the idea of physical illness even more firmly in the patient's mind and thus rendering subsequent psychotherapeutic treatment more difficult.

Psychodynamically, hypochondriasis may be regarded as a manifestation of anxiety of depression rooted in psychological conflict, social—maladjustment or a chronically low self-esteem (Sullivan, 1956). It appears that as the individual's interpersonal relationships disintigrate and his external interests constrict, his attention turns increasingly inwards, producing an acute (almost painful) awareness of hitherto unnoticed somatic sensations. The role of the central reticular formation, which normally asserts a selective blocking action on the large number of (superflous) somatic, particularly visceral, sensations jostling for attention, and thereby prevents their intrusion into consciousness, offers an intriguing neurophysiological explanation of this phenomenon. It may be postulated that a "defect" in this "filter mechanism", possibly mediated through cortical influences (via the feedback circuits now believed to exist) upon the reticular formation, may result in this morbidly increased awareness of bodily activities.

The precise nosological status of hypochondriasis is still not settled, and in most text-books it exists as a 'non diagnosis'. The American Psychiatric Association classification exalts it to the status of an independent diagnostic entity, but many authors continue to regard it as a symptom-complex which may dominate the clinical picture in a variety of psychiatric conditions—hysteria, anxiety states, obsessional neurosis, depression, schiophrenia and organic brain syndromes. The older terms such as neuresthenia and psychasthenia are now included under this heading, as also much of what Freud described actual neuroses.

Hypochondriasis is comparatively easy to recognise clinically, but the exploration of the underlying psychopathology often tax the physician's patience. Treatment would depend on the aetiologic diagnosis, and the possibility of schizophrenia must be borne in mind (Lades, 1966; Mayer-Gross et al. 1969; Kaplan, 1971; Chrzanowsky, 1959). Hoch and Polatin (1949) have described a chronic condition characterised by pan-anxiety, pan neuroses and pan-sexuality which they designate pseudoneurotic schizophrenia; this variety of psychosis is more common than many people think. Sexual disorders (impotence, loss of libido, homosexuality) are fairly common in schizophrenia (Goel, 1968a; Stekel, 1959) as well as in depression. Many western observers (Carstairs, 1961; Koestler, 1961) have noted the morbid Indian concern with sex and spermatorrhoea, which has been emphasised in the great Ayurvedic samhitas and erotic texts of ancient Hinduism (Charaka Samhita; Kama Sutra); even Mahatma Gandhi (quoted by Louis Fischer, 1951) has extolled the virtues of conservation of semen ("virya"). Goel (1968b) has highlighted the significant role played by this sexual superstition in the psychogenesis of impotence among Indian males, and D'Netto and Goel (1972) have commented on its incidence among our troops.

In treatment psychotherapy, tranquillisers, antidepressants, MIT and ECT (in some cases) have all been usefully employed, the exact choice being determined by the individual circumstances of each case. The broad principles of drug treatment would be enumerated in a later section, but explanation of body-mind relationship and the modality of drug-action is an essential prerequisite of therapy.

(ii) Psychophysiological Disorders : Better known as psychosomatic disorders, these imply an actual somatic pathology or dysfunction which is caused, at least partly, by psychological factors. While the physiological concomitants of acute emotional reactions seldom lead to a lasting somatic pathology, chronic or recurrent emotional states (anxiety, anger, frustration, depression) produce a lasting disturbance of physiological function (mediated through the limbic system and associated neuroendocrine mechanisms, the Papez Circuit, 1937) resulting in organic disease of vulnerable organ systems in predisposed individuals. It may be viewed as a failure in adaptation, a decomposition of psychological defense mechanisms which normally protect the age against intolerable anxiety resulting from intra-psychic or interpersonal conflicts. Therapy aims at resolution of the underlying conflict, strengthening of healthy defense-adaptive mechanisms, and restoration of homeostatic equilibrium. Tranquillisers and antidepressants are invaluable in providing symptomatic relief and thereby clearing the decks for psycho-therapeutic intervention.

Psychophysiolgical disorders may involve almost every system in the body : cardiovascular (psychosomatic cardiac reactions, essential hypertension, vasomotor syncope, ischaemic heart disease), gastrointestinal (dyspepsia, dysphagia, peptic ulcer; ulcerative colitis, diarrhoea, constipation), endocrine (thyroid disturbance, diabetes mellitus), obstetric & gynaecological (menstrual disturbance, sterility, difficult labour, disturbed lactation), dermatological (pruritis, eczema, alopecia and other dermatoses), and musculoskeletal disorders (backache, rheumatoid arthritis).

Most physicians are well conversant with this group of diseases, as also with the aetiologic significance of tension and anxiety in serious conditions like ischaemic heart disease. While the treatment of the organic pathology must take precedence, the underlying psychiatric pathology must be dealt with to break the vicious cycle of tension-somatic dysfunction-more tension, and effect a lasting cure. It needs to be emphasised here that the patient himself may be unaware of the psychological factors involved, and may in fact resist psychotherapeutic exploration.

(iii) "Functional overlay" has been a perpetual red herring of clinical practice, and still carries a stigma attached to it. It must however be a rare patient indeed who is not worried and concerned about his or her illness (though the degree of such concern would show individual variation, depending upon the patient's personality profile), and who does not manifest the physiological concomitants of this anxiety or depression. Merely the presence of these secondary psychological features does not justify a psychiatric referral, and should not be allowed to obscure the basic organic pathology. It should not be forgotten that even a hysteric may develop a (possibly serious) physical illness and his symptoms should not be ignored just because he "makes too much noise and creates a fuss". The skilled clinician is well equiped to deal with this psychological problem, and is in fact much better placed than the psychiatrist (who has little to do with actual treatment of the case) for undertaking supportive psychotherapy. This would apply as well to those who are being prepared for a major surgical or investigative procedure : a few words of explanation and cheer from the specialist concerned are worth their weight in gold.

CASE REPORTS

Case No. 8 : A 21 year old Gurkha soldier, was admitted to an Air Force Hospital with the h/o fever, left sided hemiplegia and aphasia of sudden onset. He was placed on DIL and transferred to a neurological centre where he continued to be on DIL while various specialised investigations were undertaken. A few weeks later, however, an alert medical specialist detected an apparent illogicality in the clinical picture: the patient, a right handed individual had left hemiplegia with aphasia: the patient was referred for psychiatric evaluation which revealed a significant feature-the individual was 'crippled" by paralysis but he himself seemed to be quite unconcerned about it and showed "la belle" indifference. His hemiplegia evaporated under narcoanalysis and the psychopathology became clear : he had been very worried about a ('forced') blood donation made some days earlier as he had been told that it damaged the brain; when he developed he became panicky. The paralysis resulted. He recovered with treatment and returned to service.

Case No. 9 : A young sepoy was referred to the psychiatrist for aphonia of sudden onset for which the ENT specialist did not find any organic basis. Pt. started talking under amytal and reveald that he had been prevented from performing "aarti" etc. to Lord Krishna in the unit which consisted mainly of people from another religion. He used to do this on the sly, behind the langar, but one day an NCO caught him at it and shouted at him; patient suddenly discovered he could not speak. He recovered with psychotherapeutic help and continued in service.

Case No. 10 : A 20 year old Armd. Corps Swr. developed sudden blindness after jumping down from his tank; the eye specialist felt there was no organic basis for his symptom. The patient required three narcoanalysis sessions before he recovered his vision and the psychopathology was unmasked; he had an immature, hystrionic type of personality and felt homesick. In addition he had severe guilt feelings regarding masturbation and had heard that it lead to blindness. Though he made a symptomatic recovery, his poor personality profile rendered him of poor military value and he was invalided out. Later it was learnt that two more persons from his unit had developed blindness after jumping down from the same tank.

Case No. 11 : A CRP jawan in his thirties presented with a long h/o changing somatic symptoms over the past 6-7 years. He had undergone lapratomy for these a year earlier but his symptoms persisted, and in addition he had developed an incisional hernia. Detailed psychiatric examination revealed free-floating anxiety, marked hypochondriacal concern (particularly relating to the genitalia), wooly thinking, somatic delusions and social constriction He improved with tranquillisers, antidepressants and ECT. His hernia was later repaired.

Cass No. 12 : A 34 year old infantryman with nearly fifteen years had seen many military and civil doctors during the previous 2-3 years for his persistent abdominal pain and sour eructations. He was finally referred to a psychiatric centre as a case of "functional dyspepsia". He had an anxious type of personality and worried about his domestic/personal problems, but had no motivation for a hysterical illness. Barium study confirmed the existance of a duodenal ulcer and he responded to conservative management combined with psychiatric treatment. It appeared that his worrying nature had probably contributed to the ulcer, and the consequent anxiety had triggered off a vicious circle.

III. Unusual and "borderline" Psychiatric conditions : Included in this group are two unusual, but by no means rare, conditions which are now arousing considerable clinical interest. The third, malingering, can scarcely be termed a medical (or psychiatric) problem, but is important for the service doctor as it often enters the scope of differential diagnosis in the broader sense.

(i) **Accident proneness :** Accidents, by definition, occur unexpectedly and by chance (or a chance-error), and it is possible that even a normal individual may have more than his share of mischance. In the latter case, however, a closer psychiatric scrutiny is warranted, particularly if the accident pattern and frequency is significantly more than the statistical probability and is associated with other features of emotional stress. As military equipment and techniques become more complex, the soldier would have to have a much higher degree of alertness, reflex-reactions and visuomotor coordination, and the results of even a minor accident could be potentially disastrous. An alert RMO could recognise the accident-prone individual and refer him for psychiatric management early, thereby averting a great deal in material cost and human suffering.

This is a relatively new field of psychiatric research and the available data is inadequate for making a definitive formulation, but among the psychological factors implicated are emotional instability, impulsiveness, relative incapacity to control hostility, egocentricity or excessive social centeredness, marked preoccupation with fantasy, feelings of insecurity (fear of loss of love or support), decreased tolerance to tension and poor vocational adjustment (Conger & Gaskill, 1971). It is possible that the psychophysiological consequences of these emotional states lead to an impairment of cognitive functions and visuomotor coordination. In a small minority of cases there may be actual evidence of minimally disordered brain function, perhaps a subictal disturbance in electrical activity recognisable in detailed EEG recordings, akin to the so-called sub-ictal neuroses. Small doses of anti-epileptic medication (diazepam, sodium dilantin) may be effective in these cases.

(ii) **Self-inflicted disorders :** Attempted suicide is the most dramatic among the many self-inflicted entities which confront the clinician, sometimes posing a difficult and perplexing diagnostic problem. The more bizaree ones involve grostesque self-injury such as self-castration (Bhaskaran, 1959) and self amuptation, often to propitiate a god or goddess. Less drastic conditions like hair-pulling, habitual scratching and skin lacerations (e. g. dermatitis artefacta) are more common (Gupta & Thacore, 1972), and may simulate other diseases. Careful investigation is needed to establish the diagnosis and psychopathology; the possibility of schizophrenia should be borne in mind, and some cases may show personality disorder of the sociopathic type.

Meninger (1938) has drawn attention to another, masked form of self-destructive acts which are aptly termed chronic latent suicide. Thus drug-dependence, alcoholism, obesity (with compulsive overeating) and cigarette smoking (with its attendent risk of cardiovascular and respiratory disease) may be regarded as indirect (sub-conscious) ways of destroying one's self, physically as well as spiritually (Kaplan, 1971). That some of these are socially accepted, if not socially-accepted and encouraged, cannot obscure the fact that many of these people have deep seated emotional problems and require psychotherapeutic help.

(iii) **Malingering** : The individual who consciously feigns illness in order to gain some selfish end or escape a difficult situation is, strictly speaking, an administrative (or disciplinary) rather than a medical problem and should ordinarily be dealt with at that level. Unfortunately, however, this individual does enter the domain of the physician who has to differentiate him from the really sick, and then decide what to do with him. The former is often a difficult undertaking (with the everpresent risk of mistaking a seriously ill patient for a malingerer—with calamitous results), and the latter nearly always poses a painful ethical dilemma. The popular estimates of its incidence in the Armed Forces appear to be highly exaggerated; it is interesting to note that the history of the Indian Armed Forces in World War II asserts that true malingering was rare among Indian troops.

Isolated episodes of malingering are of little significance, but a persistant pattern of malingering in an individual, coupled with lack of insight, inability to learn from experience and shallow emotionality, denote underlying sociopathic personality disorder; the possibility of a simple schizophrenia should also be kept in mind.

CASE REPORTS

Case No. 13 : A young Sikh soldier was hospitalised with a peculiar skin condition which defied diagnosis and did not respond to any treatment. The puzzle was solved when some cigarettes and match-boxes were discovered from under his mattress, and it was established that he had been burning his skin with lighted butts. Psychiatric examination revealed an immature personality with hystrionic and sociopathic traits, poorly motivated for service.

(The author is grateful to Capt PN Arora, Dermatologist at 92 Base Hospital for referring this case of dermatitis artefacta.)

Case No. 14 : A learned professor of medicine continued to drink and smoke heavily, leading a very intemperate life and neglecting his diet, though he was a hypertensive and had an enlarged liver. He ignored repeated friendly advice and warnings, did not take medicines regularly and seemed to be bent upon destroying himself. Psychiatric inquiry revealed severe emotional problems, particularly in relation to his marital life (he was estranged from his wife, largely due to the harmful influence of his domineering mother) but he did not cooperate in subsequent treatment. A year later he suffered a massive stroke which left him a physical and mental cripple-hemiplegic, aphasic and demented. A consultant neurologist (who was unaware of the psychological background) remarked that the patient seemed to lack the will to recover !

Case No. 15 : A brilliant young doctor, only child of a famous educationist (who was himself a widower) had taken to pethidine while in the A. M. C., and was invalided out of service. Subsequently he came under the author's treatment at a private psychiatric hospital; at the time of admission he was taking 40 ampoules of pethidine daily. Intensive psychotherapy revealed that his addiction represented an attempt to "lose" himself, the result of an extended mourning reaction for his beloved, and suppressed (oedipal) hostility towards his father. He had deeply loved his mother and her untimely death had left a permanent scar on his young mind. Later, as an adult, he fell in love with a fellow medical student and they were informally engaged. The girl, however, died suddenly due to a preexistant illness, leaving him stunned with grief at this loss of yet another love-object (for which, through a complex subconscious process, he blamed his father). Around this time he had an attack of "renal-colic" (which may have been psychosomatic pain) and was administered pethidine. The ensuing relief from pain and anxiety showed him the easy way out of his emotional distress, and he feigned illness to get more of the same. He continued to take the drug (which he found easy to procure, being an M. O.) after discharge from hospital and slipped into addiction. He seemed to recover and had been drug-free for over eight weeks at the time of leaving hospital. He returned home to join a post-graduate course in ophthalmology, but relapsed within a few months and was rehospitalised, again with 40 amps. pethidine daily.

TRANQUILLISERS AND ANTIDEPRESSANTS IN THERAPY

Since many of the conditions described in earlier sections would ordinarily be treated by physicians and surgeons, rather than by a psychiatrist, it would not be out of place to deal briefly with the drugs used in their managent. It may be emphasised here that adequate doses for sufficiently long periods should be used; there is no justification for employing homeopathic doses for brief periods and then giving up the drug as ineffective. A variety of major and minor tranquillisers and antidepressants are now available in the PVMS.

Minor Tranquillisers : So called because they are used mainly in the treatment of neuroses, PV 01771 Chlordiazepoxide (Librium) 10 mg tablets and PN 01780 Diazepam (Calmpose) 5 mg tablets are safe and effective in a broad spectrum of nonpsychotic conditions. In addition to relieving anxiety they also act as mild antidepressants, and are usually employed in dosage of 1 tab tid or qid. There is no significant sedative effect or impairment of cognitive functions and the patient may remain on his job. Both these drugs have mild anti-convulsant activity as well, and potentiate the effects of other antiepileptic medication. For this reason they are useful also in cases of head injury, effects of.

Major tranquillisers : Introduced into psychiatry by Delay et al nearly two decades back, these belong almost exclusively to the growing phenothiazene family. PV 01386 Trifluoperazine (Eskazine) 5 mg tab and PV 01778 Chlorpromazine (Largactil) 25 mg tab are employed primarily in the treatment of psychoses, but smaller doses (Eskazine ½ tab b. i. d. or t. i. d., or Largactil 1 tab b. i. d. or t. i. d.) can be usefully employed in variety of nonpsychotic conditions as well particularly in hypochondriacal and psychophysiological syndromes involving GIT symptomatology. Much higher dosage (e. g. upto 600-1200 mg of Largactil daily) are used in psychotics. Postural hypotension and extrapyramidal side-effects are uncommon with smaller doses, and the latter is readily amenable to PV 01769 Trihexyl Phenidyn Hydrochloride (Pacetane) 2 mg tab, 1-2 tabs daily. True hepatotoxicity is rare. PV 01860 Thioproperazine (Majeptil) 5 mg tab is used almost exclusively in major psychoses.

Antidepressants : Two tricyclic compounds, PV 0178 Imipramine (Tofranil) 25 mg tab and Amitryptilline (Sarotena) 25 mg tab, are available and can be used alone or combined with one of the tranquillisers; Sarotena itself has an additional anxyolytic effect. The usual dosage is 1-2 tabs t. i. d., and the optimum effect of the drug is apparent after 2-3 weeks of continuous therapy. It is therfore unwise to give up therapy after only a few days and conclude that the drug is ineffective. These drugs have atropine-like side effects (dryness of mouth, tachycardia, blurring of vision, constipation) but they are seldom severe and subside after a few days. There may be postural hypotension in some patients.

SUMMARY

The concept of psychogenicity in medicine is reviewed in its historical context and a functional classifications for the many somatic manifestations of psychiatric illnes is proposed. These conditions are discussed in their theoretic, clinical and therapeutic aspects, and the criteria for diagnosis are restated precisely. Illustrative case reports have been appended and the role of drugs in management is briefly enumerated.

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Nurtured by experience and critical review, ideas evolve into concepts and provide new insights. The theme of this paper gradually emerged during three memorable years in Lucknow under the guidance of Surg. Capt. T. B. D' Netto, I. N., Senior Adviser in psychiatry and to him the author owes a lasting debt of gratitude. Thanks are due also to Col. A. Jayachandran, AVSM, Officer Commanding, 148 Base Hospital, for his encouragement and for the permission to publish this paper.

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EVALUATION OF TREATMENT IN NASAL ALLERGY†

BY

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INTRODUCTION

Nasal Allergy is a common disorder encountered in clinical practice. The condition is not inconsistent with normal life but is so distressing at times that it renders the individual miserable in social and domestic circles. Besides specific desensitisation, various non-specific measures have been tried from time to time to treat this common disorder.

The present study was undertaken to evaluate the relative merits of the nonspecific modes of treatment, carried out for nasal allergy in routine ENT practice.

MATERIAL AND METHOD

The cases were selected from ENT out patient department of Command Hospital, Pune for a period from June 1971 to May 1972. The diagnosis of nasal allergy was based on the clinical picture :

(a) The presence of three main symptoms (i) watery rhinnorrhoea (ii) nasal obstruction, and (c) paroxysmal sneezing.

(b) The presence of local signs in the form of oedema of turbinates, pallor of nasal mucosa and watery discharge.

The selected cases were then divided at random into four groups of 20 each and categorized exclusively for one of the following lines of treatment :—

GROUP I—Oral Antihistamines and Local nasal decongestant drops

Tablet Pheneramine Maleate 25 mg (Avil) orally at 12 hourly intervals and 1%

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ephedrine in normal saline nasal drops locally three times a day were administered to this group for a period of 4 to 12 weeks depending upon their response to the therapy.

GROUP II—Zinc ionisation therapy

This group was treated by Zinc ionisation therapy twice a week for four weeks. The exact mechanism of action is not clearly understood. However, the following postulations have been made :—

(a) Zinc ion reacts with receptors in the walls of the blood vessels on which 5 hydroxytryptamine acts and thereby it acts as a competitive antagonist.

(b) Zinc ion is deposited on the surface of the nasal mucous membrane as a result of chemical reaction. It makes the mucosa less sensitive to the offending agent.

(c) It alters the pH of the nasal secretions which brings about shrinkage of the nasal mucosa.

GROUP III—Electrocauterization of inferior turbinates

The third group was treated with electrocauterization of inferior turbinates. A broad straight point of the galvano cautery was used and entire length of the inferior turbinate was cauterized with red hot cautery. The galvanocautery destroys the soft tissues. The total reaction produced stimulates fibrosis which makes the tissue less sensitive. The patency of nasal airways is at the same time improved.

GROUP IV—Submucosal intranasal corticosteroids

The fourth group was treated with submucosal injections of 12.5 mg (0.5 cc) of hydrocortisone acetate in either inferior turbinate. Dental syringe with a long thin needle was used for this purpose and a total of 4 such injections were given at weekly interval.

ASSESSMENT OF THERAPY

The patients under treatment were reviewed at weekly intervals for a period of 6-8 months. Any relief in subjective symptoms was noted. The nasal cavity was examined for any reduction of oedema, size of the turbinates and nasal secretions.

Based on the above observations, the results were expressed as under :

Good

(a) Complete relief of symptoms

(b) Complete disappearance of oedema and watery nasal discharge

(c) Nasal mucosa returning to normal color

(d) Appreciable improvement in histological picture.

Satisfactory

(a) Mild symptoms persisted but the patient markedly relieved.

(b) Mild oedema and watery discharge persisted.

(c) Partial improvement in post therapy histopathology.

Poor

No improvement whatsoever.

OBSERVATIONS AND DISCUSSION

Group I—Oral Antihistamines and local nasal decongestant drops

The most satisfactory response to the antihistaminics has been noted in patients with seasonal allergy.

In the present series of 20 cases of perennial nasal allergy, treated with 25 mg pheneramine Maleate (Avil) twice a day for a period of 4 to 12 weeks, the overall success was noticed in 55% cases. Reading and Malcomson (1954) reported satisfactory response in 69.6% cases from the use of anti-histaminics. Hansel F. K. reported symptomatic relief in 80% cases with oral administration of tridecamine dospen, a tablet containing 20 mg each of Deoxylamine Succinate, phencramine Maleate and pyrilamine Maleate at 8 to 12 hourly interval. The cases treated in his series were mostly belonging to seasonal allergy. Edmonds (1959) reported beneficial results in 87% cases with oral administration of Parabrondylamine Maleate (Dimetane) tablets 12 mg at 8 to 12 hourly interval in cases of seasonal and perennial allergy.

The lower percentage of success in the present series is attributed firstly, to the perennial nature of the condition treated, secondly to the smaller dosage of the drug administered and thirdly to the presence of polyposis and hypertrophy in 20% of the cases under treatment.

The duration of allergy also had an effect on the results. This therapy was effective in 75% cases where a duration of allergy was less than 2 years, whereas it was effective only in 25% cases where the duration happened to be longer. Reading and Malcomson (1954) and Hansel F K (1959) made similar observations while treating cases of nasal allergy with antihistaminic drugs.

Sneezing and rhinorrhoes were relieved in 60% and 55% cases respectively, whereas nasal obstruction was relievd only in 42% cases. This finding is consistent with the findings of Williams D Mackee (1968) who said that, antihistamines are particularly helpful in controlling sneezing and rhinorrhoea though not nasal obstruction to a similar extent.

None of our cases who had polyposis or hypertrophy of inferior turbinates showed improvement with this therapy. Talor L (1966) in describing treatment of perennial nasal allergy had made a similar observation.

Decongestant drops in the form of 1% Ephedrine in isotonic saline produced symptomatic relief by causing shrinkage of the mucosa. No adverse effect of the nature of rhinitis medicamentosa or rebound congestion was noted.

Group II—Intranasal Zinc Ionization

In this series of 20 cases treated with zinc ionization, 75% showed considerable improvement during the follow up period of 6 to 9 months. Baily & Shields (1937) claimed complete relief of symptoms in 54.6% and considerable relief in 36.0% of nasal allergy. Gray (1961) estimated an overall success rate of 56% initially, falling to 25% over a period of one year. More recently, Wair C. D. (1967) reported 61.4%complete relief, 24.8% improvement and 13.6% failure, one month after the therapy in his series. In long term assessment, the suceess rate was 43.1%. The results obtained in the series under review were consistent with the results obtained by the above mentioed workers.

This mode of treatment has been effective in controlling all the symptoms to the same extent. Hypertrophy of the turbinates did not show any appreciable reduction after the completion of therapy. Reversal of histological changes was observed in 58.8% of cases. No adverse effects were noticed during the course of the therapy.

Group III—Electrocauterisation of Inferior Turbinates

In this group, improvement was noticed in 70% of the cases in the present series 6 (85.7%) of 7 cases who had marked hypertrophy of inferior turbinates showed complete relief of symptoms thereby showing that this treatment was more effective in the cases of nasal allergy associated with hypertrophy of the turbinates and with prominent complaint of nasal obstruction The therapy was combined with submucosal resection of the septum in 3 cases because of associated deflections of the nasal septum. According to Hiranandani N L (1966) galvanocauterisation of inferior turbinates has given fairly good results in selected cases of allergic rhinitis. Simpsom and Groves (1958) also reported good results with this therapy.

Group IV—Intranasal Submucosal Corticosteroids

The last group consisted of those cases where submucosal infiltration of corticosteroids was the mode of treatment. The exact mechanism by which local infiltration of corticosteroids helps in the nasal allergy is not known, but it is thought to suppress the trigger zones in the nasal cavities.

In the present series of 20 cases, success was achieved in 85% cases. Those cases were followed upto a period of 9 months. Wall & Shure (1952) reported 80% success for a period from 6 to 10 months by injecting submucosal cortisone. Sidi & Tardiff (quoted by Baker & Strauss 1962) got 60% results with hydrocortisone acetate. Simmons (1960) obtained relief in 75% cases with this mode of treatment. Mawat (1961) treated cases of allergic rhinitis with submucosal injections of hydrocortisone and 88% cases showed definite improvment. Baker and Strauss (1962) treated cases of nasal allergy with 3 to 4 injections of submucosal corticosteroids at weekly intervals and recorded 75% success rate. Gill (1960) reported 81.25% results with dexamtehasone and 84.8% with predinisolone acetate. Sardana et al. (1969) achieved 75% success with hydrocortisone acetate. The results achieved in the present series are consistent with the results obtained by above workers.

This therapy has been found effective in controling all the symptoms of nasal allergy. There was marked reduction in the size of oedematous turbinates in 17 (94.4%) of the 18 cases. Out of the two cases of nasal polyposis, one showed definite reduction in size on completion of treatment. This is consistent with the findings of Myers (1958) who reported beneficial result with intrapolyp injections of corticosteroids.

The intranasal injection was not painful and as a rule, treatment was tolerated without discomfort to the patient. In almost all but 3 cases relief was recorded after the first injection and was maintained in the subsequent injection.

There was no systemic effect from intranasal injection of hydrocortisone acetate as the quantity injected was very small in comparison with oral or pararitral drug used to get similar local benefit. There was no untoward reaction in any case from this mode of treatment. Those observations were consistent with those made by Baker & Strauss (1962).

CONCLUSIONS

80 cases of perennial nasal allergy were studied in an attempt to evaluate nonspecific treatment in nasal allergy. Intranasal submucosal hydrocortisone acetate was found to be the most effective nonspecific treatment in cases of perennial nasal allergy. This gave an overall success of 85% during the follow up period of 6 to 9 months.

Of the 20 cases 12(10%)were completely relieved of symptoms 5(25%) were considerably relieved whereas only 3 (15%) failed to respond to the treatment. The injections were painless and easy to be administered. No untoward reaction was noticed. Hypertrophic turbinates and polyp also showed reduction in size with this mode of treatment.

Intranasal zinc ionisation was found to be the second best mode of none specific treatment. 75% was the overall success rate with this therapy. 10 (50%) enjoyed complete relief of symptoms for a period of upto 9 months, 5 (20%) showed satisfactory response whereas in 5 (25%) cases, it failed to produce any beneficial results,

Electrocauterization of inferior turbinates was found to be very effective in cases who had associated hypertrophy of the inferior turbinates and the prominant complaint was of nasal obstruction. The overall success rate with this mode of treatment was 70%. 8 (40%) had complete relief upto a period of 9 months, 6 (30%) had satisfactory response and 6 (30%) failed to show any improvement.

The treatment with oral antihistamines and local decongestant drops was effective only in 55% cases. 4 (20%) enjoyed complete relief of symptoms for a period of 6 months while 7 (35%) were considerably relieved. The treatment was a complete failure in 9 (45%) cases. This mode of treatment was found to be more effective in the cases with shorter duration of symptoms.

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ENDRIN POISONING

(A case report)

BY

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Endrin is one of the chlorinated insecticides used as horticultural sprays for pears, apples, cherry trees and black current bushes. The pesticides may be toxic not only to lower forms of life but also in varying degrees to the higher animals and to man. When present in our foods or ingested accidently, they may cause injury to health. Two cases of accidental poisoning with Endrin are reported here.

CASE REPORT

Case No 1 : A female aged 42 yrs. was admitted in Command Hospital (Central Command) Lucknow on 6-12-68 at 12.00 hrs. with the history of having accidently ingested ½ Teaspoonful of Endrin thinking it to be cough linctus.

Examination on admission showed that the patient was in semiconscious state. She was getting retching and epileptiform convulsions occuring at abaut 15 to 20 minutes interval. There was no neck rigidity. Both pupils were constricted and sluggishly reacting to light. Pulse 110/mt. BP 130/80 mm of Hg. CVS and resp. sys. NAD.

INVESTIGATIONS

(a) Blood TLC 15, 200/cumm. P 95, L 4, E 1%
Hb 10. 5 Gm%
BT 2 mts 5 sec.
CT 4 mts 25 sec.

(b) Urine Reaction-acid. Albumin present (+) Sugar +.
Chloride. 6 Gm/lit. Deposit—NAD.

(c) Blood Sugar 106 mgm%

(d) Liver Function Tests within normal limits.

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(e) Blood Urea 20 mgm%

(f) Serum electrolytes. Na 130 meq/lit. K 4.0 meq/lit Chloride 100 meq/lit. CO_2 12 meq/lit

(g) S G O T 32 I U/lit. S G P T 5 IU/lit.

(h) Urine culture. Moderate growth of proteus morgani. Sensitive to polymyxin only.

MANAGEMENT AND PROGRESS

Patient was immediately given gastric lavage with large volume of water. She was also given Inj. phenobarbitone 400 mgm. IV; Calcium gluconate 10 ml 10% IV; Inj. streptomycin 1 Gm IM and 5% glucose drip. Her convulsions could not be controlled with the above therapy as such a pentothal drip was started which acted favourably.

On next day morning she started regaining her consciousness. She was put on reverin IV 275 mgm B.D; Inj. Neurobion 3 ml 1 M. O D. and Inj. B. complex 2 ml IM O D. On 18-12-68 i. e. after 48 hrs. of admission she became fully conscious and started accepting oral feeds. Her further recovery was uneventful and was discharged on 23-12-68.

Case No 2 : A male aged 22 yrs. was admitted to Command Hospital (Central Command) Lucknow on 6-12-68 at 12.00 hrs. with the history of having ingested accidently about 2 Teaspoonful of Endrin. Patient was in unconscious state. He vomited thrice in the M I Room and was getting continuous epileptiform convulsions. Attacks were coming at the interval of 1-2 mts. There was no neck rigidity. Pupils — constricted and sluggishly reacting to light. Pulse 140/mt. BP 130/50 mm of Hg. C V S and Resp. sys. N.A.D.

INVESTIGATIONS

(a) Blood : TLC 12800/cumm P 94. L5. E 1%. ESR 32 mm fall lst hr.
Hb 14.0 gm% BT 1 mt 50 sec. C. T. 4 mts 10 sec.

(b) Urine : Reaction acid. Albumin present (++) Sugar absent. Chloride 4 Gm/Lit. Deposit showed numerous R B C's. Puscells 4-6.
PHF with few epithelial cells.
Specific Gravity **10 12.**

(c) Liver Function Test within normal limits.

(d) S G OT. 98 IU/lit. S G P T. 29 IU/lit.

(e) Blood Urea 55 mgm%.

(f) Serum electrolytes. Na 120 meq/lit. K 5. 0/meq/lits. Chloride 100 meq/lit. CO_2 10 meq/lit.

(g) Urine culture. Moderate growth of proteus morgani sensitve to polymyxin only.

(h) E.C.G.—within normal limits

MANAGEMENT AND PROGRESS

Patient was immediately given the following treatment. Gastric lavage with large volume of water. Inj. phenobarbital 400 mgm IV; Inj. paraldehyde 5 ml deep IM, Atropine 1/100 gr IV; Inj. streptomycin 1 gm lm; Inj. cal. gluconate 10 ml 10% IV. and 5% glucose drip along with soda bicarb 7.5% 40 ml. His convulsions could not be controlled with the above treatment as such he was put on pentothal drip and was given an injection of Flexedil. Next day he was put on Inj. Reverin 275 mgm IV. B D; 50% glucose 50 ml IV 6 hrly. Inj. Neurobion 3 ml IM OD, Inj. Durabolin 25 mgm IM. and Phenobarbitone 200 mgm IM. stat.

After 48 hrs. of admission patient exhibited signs of anuria. He had passed only 85 ml of urine during the previous 24 hrs. (catheter specimen). He was still unconscious and was getting occasional abnormal movements. He also had epistaxis and his gastric aspirate was blood stained. His breathing became laboured though lungs were clinically clear. Temp 101°F. Blood Urea 60 mgm%. The above line of treatment continued.

On 3rd morning patient started regaining his consciousness and his urine output increased to 200 ml/24 hrs. Lungs-clear. Temp. 102°F. Inj. Largactil 25 mgm 1M OD and Inj. siquil 10 mgm IM BD were added to the above line of treatment. On 4th day he became fully conscious and started accepting oral feeds. His blood urea went upto 111 mgm% and CO_2 combining power remained at 13 meq/ litre. Hence he was given 50% glucose 500 ml and 25% glucose 750 ml in IV drip with sodabicarb 7.5%-40 ml. Urine output increased to 800 ml/24 hrs. and he showed improvement during the subsequent 4-5 days.

On 9th day of admission he started running high fever, looked toxic and his blood urea went upto 222 mgm%. Respirations were acidotic type but lungs remained clear. Blood culture revealed moderate growth of staphylococcus pyogenes, sensitive to penicillin only, while urine culture indicated moderate growth of klebsiela aerogenosa, sensitive to polymyxin and furadantin. ECG showed tall and peaked T waves in V^1-V5, patient was put to cap ampicillin (250 mgm) 2 caps 6 hrly; furadantin. 1 Tab BD; along with vitamins and high carbohydrate diet.

On 11th day he started having diuresis. Fever was still continuing. He had pitting oedema over the left leg. ECG showed inverted T in lead V1-V3 and reduced height of T in V5-V6 suggestive of hypokalaemia. He was given I V drip with 1.5 gm of pot chloride and orally pot. chloride 2 gm TDS with soda bicarb 2 gm TDS and sod. chloride 2 gm TDS along with the antibiotics.

On the 15th day the patient developed an abscess on the left leg which was followed by an abscess on left arm. He also developed signs of pneumonia which was confirmed on X' ray. Blood culture then showed a heavy growth of staphylococcus pyogenes. This staphylococcal pyaemia was treated adequately with penicillin, erythromycin, and chloromycetin and the patient showed continuous improvement and was eventually discharged from hospital after 3½ months of total hospitalisation.

DISCUSSION

Organo-chlorine compounds act as central nervous system poisons though the mechanism of this action is not known. Most of the cases occur due to accidental ingestion of the poison. Industrial absorption is commonly through skin. The main members of this group in common use are Endrin, chlordane, heptachlor, dieldrin and diendrin. The chemical name given to these compounds is Haxachloro octahydroendo endodimechano nephthalene. Endrin is the epoxide derivative of isodrin and also diastereo isomer of dieldrin.

These are fat soluble, highly stable insecticides used as dusts, wetting powders and solutions in organic solvents. Most toxic of the group is Aldrin. Skin absorption of Indan derivatives in organic solvents may be fatal within one hour. These compounds are deposited in fat and once deposited are virtually not excreted even over the subjects life time. For an adult man oral dose of 10 mgm/Kg produces acute illness and 1 to 3 gm of these derivatives produces severe symptoms and may be fatal.

Endrin acts as general protoplasmic poison perhaps by virtue of its affinity for lipids. Damage is widespread and may involve central nervous system, liver, lungs, kidneys and blood vessels generally. Its most striking effect is stimulation of central nervous system such as hyperexcitability, tremors, ataxia and convulsions beginning within thirty minutes to six hours, followed by depression. In mild cases there may be giddiness, headache, nausea and twitchings. In persons exposed over a period, a condition indistinguishable from epilepsy may be presented. Even before the development of fits, ECG shows epileptiform changes. Death is usually due to respiratory failure, asphyxia during a convulsion, or renal failure. In some cases ventricular fibrillation may be the immediate cause of the fatal outcome.

Treatment consists in immediate removal of contaminated clothes, thorough skin cleansing, gastric lavage in cases of ingested poison and administration of barbiturates or sodium thiopentone. There is no specific chemical test to confirm the diagnosis, nor are any specific antidotes available. As such most of the treatment remains to be symptomatic in nature.

CONCLUSION

Two cases of accidental endrin poisoning are presented here with the similar mode of onset in both the cases. Our case No. 1 was of a mild degree poisoning and did not show any complications. Our case No. 2 who had ingested four times the dose as compared to case No. 1 went into acute renal failure indicating severe degree of poisoning. During his recovery from renal failure he developed staphylococcal pyaemia and staphylococcal pneumonia. The management and progress of both the cases have been discussed in detail. Energetic treatment instituted immediately can reduce the mortality in such cases.

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RHEUMATOID PULMONARY DISEASE

(A Case Report)

BY

Maj S.R. Bhowal,* Maj Yash Pal, and Col K.K. Gupta*****

The systemic nature of rheumatoid arthritis was first emphasized by late Dr. Philip Ellman and he ádvocated the use of the terms rheumatoid disease and rheumatoid lung.

It has been observed in patients with rheumatoid arthritis that systemic granulomatous lesions when demonstrable have a microscopic appearance similar to that of subcutaneous Rheumatoid nodules. In the absence of Pneumoconiosis such lesions of the lung are believed to be rare (Flatley F. J., 1959). Ellman and Ball (1948) and Ellmand and Cudkowiex (1955) described several cases of rheumatoid arthritis with associated lung lesions ranging from diffuse chronic fibrosing interstitial pneumonitis to a sterile empyema (Raymond Daley & Henry Miller, 1966). Fairly large number of cases of rheumatoid disease in association with involvement of the lung parenchyma have recently been reported and the histological evidence of specific rheumatoid features in the lung of at least some of the cases, makes it highly suggestive that rheumatoid fibrosing interstitial Pneumonitis is a definite entity (John Crofton & Andrew Douglas 1959). Upto 1965 Stack & Grant were able to analyse 99 reported cases. Cruickshank (1957) reported 6 cases among 100 autopsies of rheumatoid arthritis. Patterson et al. (1965) found it in 1. 1% of 702 patients.

We report here a case of rare pulmonary manifestations of rheumatoid disease, that is a case of rheumatoid diffuse chronic fibrosing interstial pneumonitis.

CASE REPORT

Patient a 62 years old lady was admitted to Command Hospital, Calcutta on 08 Dec. 72 with the complaints of pain and deformity of both large and small joints and

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gradual loss of body weight of 10 years duration, recurrent respiratory tract infection with gradually increasing breathlessness on accustomed exertion of six months duration. Pain and swelling first appeared in the left knee. Left wrist and small joints of the left hand–gradually all other large and small joints started getting involved followed by restriction of movement of flexion deformity (Fig. 1). The distribution was fairly bilaterally symmetrical. She used to have fever off and on specially along with acute episodes of joint pains.

Clinical examination revealed a poorly nourished crippled old lady, Afebrile, Pulse–82/minute regular. Patient was orthopnoic. Blood pressure 150/90 mm of Hg. No dependent pitting oedema. Slightly raised jugular venous pressure. No cyanosis or clubbing was present. There was no Lymphadenopathy, rheumatoid nodules or any evidence of vasomotor affections. Kyphosis of spine with flexion deformity hip and knee joints. Flexion deformity of the terminal interphalangeal joints with Ulnar deviation of the fingers both sides. Ankle joints had eversion deformity (Fig. 2). On examination of lungs normal vesicular breathing with crackling rales heard all over the chest and more at the interscapular region (Fig. 3). No other systemic manifestations. Investigations revealed, Blood ESR—17 mm fall in Ist hour (Wintrobe), Haemoglobin 13.0 gm/100 ml, TLC—9.800/cmm, DLC—Neutrophil 74% Lymphocyte 19% Monocyte 1°/o Eosinophil 6%. Sputum for AFB repeatedly negative. Urine showed no significant abnormality. Blood uric acid 3.5 mg°/o No. L.E. Cell phenomenon seen. Rose-waaler test DAT 1 : 32. Blood urea 35 mgm/100 ml. Serum protein electrophoresis albumin 41°/o, α1-globulin 15°/o, α2-globulin–15°/o β-globulin 18°/o, γ—globulin—26°/o, X–Ray chest shows extensive fibrosis in both lung fields with honey–combed appearance. Lesions were more in lower zones. X–Ray hands showed deformed fingers with almost disappearance of the joint spaces from wrist downwards including the fingers (Fig. 4). The bones were osteoporosed. There was atrophy of the distal phalanx of the 4th finger.

Diagnosis of Rheumatoid lung was based upon the characteristic clinical and Radiolgical picture of Interstiall pulmonary fibrosis in a case of Rheumatoid arthritis. No other aetiological factor contributing to similar type of Radiological findings could be established.

Lung biopsy could not be done because of low general condition of the patient. Pulmonary function tests revealed reduction in both, forced expiratory volume and forced vital capacity but the FEV/FVC ratio was normal-suggesting restrictive lung disease without airway obstruction.

DISCUSSION

In Rheumatoid disease articular lesions usually precede the onset of systemic manifestations as in this case.

Four different pulmonary conditions claimed to be specifically associated with Rheumatoid arthritis (Raymond Daley & Henry Miller, 1965). They are Pleural adhesions, Pleural thickening and pleural effusion, Diffuse chronic fibrosing interestitial pneumonitis, Rheumatoid pneumoconiosis (Caplan's syndrome) and Rheumatoid nodules in the lung. In addition a higher percentage of respiratory infection has been observed in patients with Rheumatoid arthritis (John Crofton & Andrew Douglas, 1969).

Progressive massive pulmonary fibrosis was once regarded as a form of tuberculosis modified by coal dust. Caplain ruled out the possibility of tuberculosis as a cause of Rheumatoid arthritis and the pulmonary lesions. He postulated that in patients with Rheumatoid arthritis, more fundamental, generalized tissue abnormality was responsible to produce the particular pulmonary reactions to dust and infection which he observed (Flatley F.U. 1959). The definite etiologic factors and their mechanism of action in the production of Rheumatoid pulmonary disease remains undetermined.

The lung lesions in Rheumatoid disease is unresponsive to antibiotic therapy unless there is superimposed bacterial infection. Corticosteroid administration is usually found to be useful in arresting the further progression of the disease process. Early management of superimposed infection with appropriate antibiotics and corticosteroid therapy at the time of exacerbation appears to be the treatment of choice of Rheumatoid pulmonary disease.

SUMMARY

A case of Rheumatoid disease is discussed with manifestations of Arthritis and Insterstial pulmonary fibrosis.

It has been stressed that in Rheumatoid arthritis with pulmonary lesions, diagnosis of Rheumatoid Pulmonary disease must be considered.

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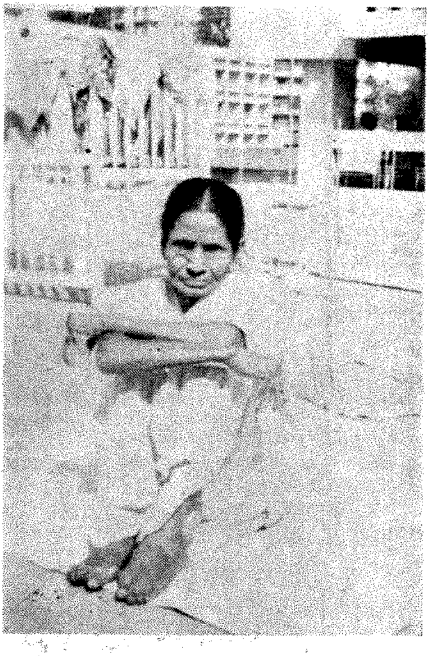

Fig. 1

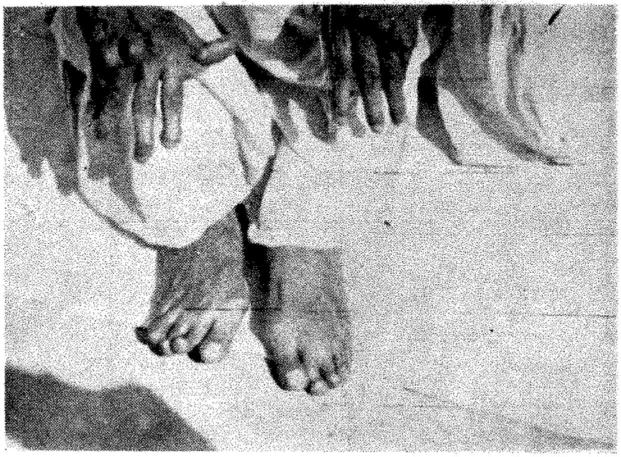

Fig. 2

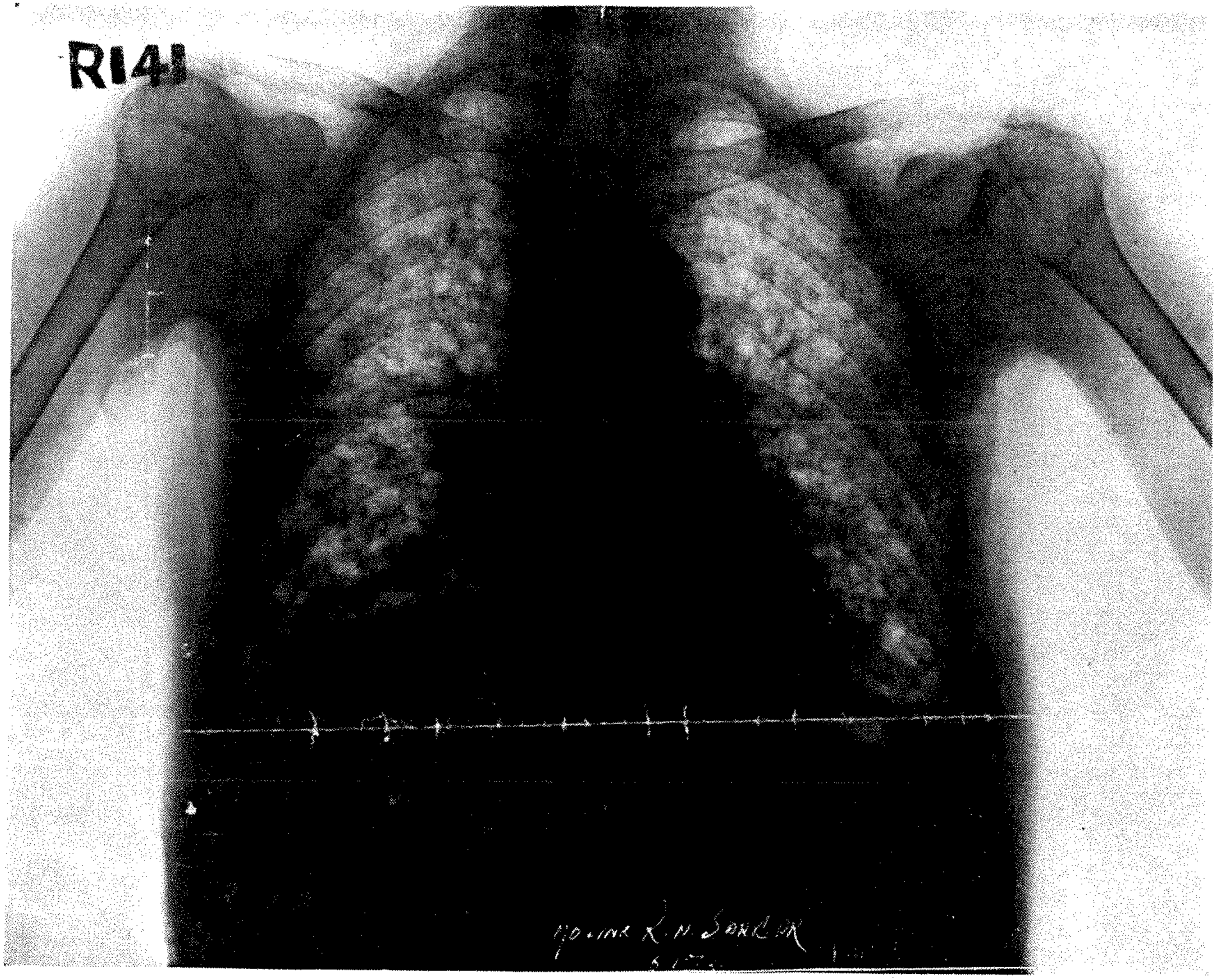

Fig. 3

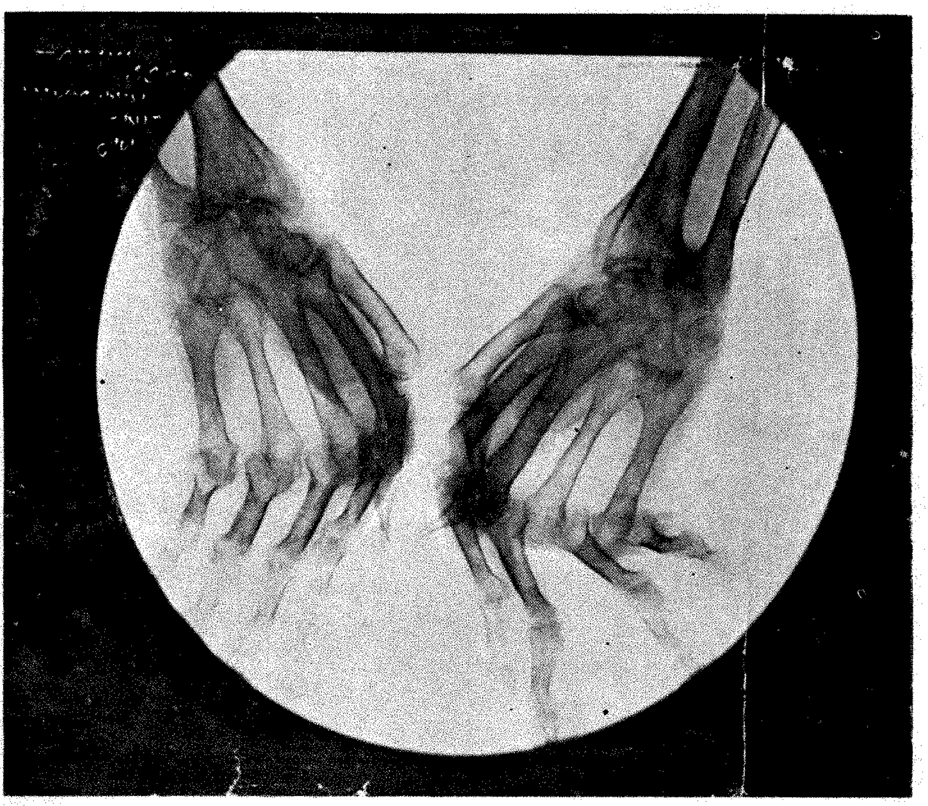

Fig. 4

ABSTRACT

IMPROVED SURVIVAL FROM ACUTE RENAL FAILURE AFTER TREATMENT WITH INTRAVENOU SESSENTIAL L-AMINO ACIDS AND CLUCOSE

Ronald M. Able et. al,

New Eng. J. Med. 288, 695, 1973

Inspite of early institution of dialysis, acute renal failure carries a very high mortality rate due to diverse causes. The use of the Giordano and Giovannetti diet regime has contributed somewhat to the reduction of mortality in acute renal failure. The use of such diets however and benefits therefrom depend upon the adequacy of gastrointestinal functions. It was therefore logical that the authors carried out a controlled trial using the same principles but the "diet" was given intravenously.

The effect of intravenous essential L-amino acids and hypertonic glucose ("renal-failure fluid") in the treatment of acute renal failure was evaluated in a prospective double-blind clinical trial. The population of 53 adult patients had sustained a presumable renal insult within 10 days before inclusion in the study. The control group was treated with glucose alone. The two groups were adequately randomized for age, renal diagnosis and renal dysfunction at the time of the study. Twenty-one of 28 patients receiving renal-failure fluid, as compared with 11 of 25 given glucose alone, recovered from these episodes of acute renal failure ($p=0.02$). These survival differences were more apparent in higher-risk patients, including those with oliguric acute renal failure, requiring dialysis and those in whom pneumonia, generalised sepsis or major gastrointestinal hemorrhage developed. Analysis of indices of renal function suggested more rapid recovery from acute renal failure in patients treated with renal-failure fluid. These data suggest that treatment of such patients with this solution significantly improves survival, which may be related to shorter overall duration of renal dysfunction.

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ABSTRACT

CORONARY ARTERIAL SPASM IN PRINZMETAL ANGINA

Oliva, P. B., Potts, D. E. & Pluss, R. G.

New Eng. J. of Med. 288, 745, 1973

In the 1950's Prinzmetal et al described a type of Angina, different in many respects from the classical Heberden's effort induced Angina. While post-mortem and coronary angiographic studies have shown organic obstruction in the classical angina, the cases of Prinzmetal-Variant have failed to show extensive or impressive coronary occlusion, constantly. For these reasons many observers have believed that the Prinzmetal type may be due to "coronary spasm", producing transient and variable obstruction.

In this paper, Oliva et. al have shown coronary vasospasm on arteriographic records. And, such spasm are noted in arteries distal to diseased one and found in normal arterial segments, in fact. The authors opine that aorto-coronary saphenous-vein by pass grafts may not be a suitable form of corrective for these cases.

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ABSTRACT

FACTORS THAT INTERFERE WITH THE MANAGEMENT OF DIABETES MELLITUS

Drug & Therapeutic Bulletin. Vol.11, No. 2, Jan 1973

Satisfactory management of diabetes mellitus requires consideration of many different aspects of patient's life. Where difficulty in control occurs important factors to bear in mind can be summarised as follows:–

I Problems of every-day life

(a) Adolescents often resent advice.
(b) Variation in work or study and exertion.
(c) Anxiety, over-work or personal affairs.
(d) Diabetes may worsen before menstrual periods and on oestrogenic contraceptives.
(e) Pregnancy - It may require a change in dosage of insulin.
(f) Consumption of alcohol can lead to unpleasant flushing in patients on sulphonylureas.
(g) Renal threshold rises with age and with renal failure.

II Intercurrent Illnesses

(a) Infections exacerbate diabetes.

(b) Endocrine disturbances affect as follows :–
Thyrotoxicosis, acromegaly, growth hormone administration, Cushing's syndrome and pheochromocytoma (increased insulin requirement); Adrenal or pituitary insufficiency (increased sensitivity to insulin).

(c) Miscellaneous–
  (i) Myocardial Infraction in older patients may precipitate ketosis.
  (ii) Liver disease or uraemia may appear to lessen the severity of diabetes.
  (iii) Gut disorders viz. Malabsorption, anorexia and dysphagia decrease blood glucose; "dumping" syndrome or neuropathic disturbance of gastric emptying can cause erratic hypoglycaemia. Portacaval shunts allow glucose to enter the circulation unchecked.

III Problems in Insulin Therapy

- (a) Dispensing errors.
- (b) Storage of insulin in warm places or deep freeze.
- (c) Inability to measure doses correctly due to poor sight, confusion in old age or leaking syringes.
- (d) A lump at the site of injection leads to irregular and uncertain absorption.
- (e) Antibodies form to most injected insulins.
- (f) Renal threshold may be high or low. Blood glucose level is a better test than urine sugar.

IV Effects of Coincident Therapy

- (a) Increase in blood glucose occurs by–
  - (i) Inadvertant intake of glucose or other carbohydrate
  - (ii) Acetazolamide and thiazide diuretics
  - (iii) Phenytoin and diazoxide
  - (iv) Corticosteroides, oestrogen, thyroid hormone, levodopa, and morphine.
- (b) Reduction in blood glucose is brought about by–
  - (i) Drug increasing insulin secretion and reducing clearance of sulphonylureas e. g. sulphonamides, salicylates, barbiturates, M. A. O.—inhibitors.
  - (ii) Prapranolol enhances hypoglycaemia when liver glycogen stores are depleted.
  - (iii) Chlorpropamide because of its prolonged action as well as induced anorexia.

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ABSTRACT

STEROID DRUGS IN CHEST DISEASES

'To-Days Drugs'

Brit. Med. J. Vol. 1, pp. 459-460, 20 May 1972

Steroids have been used in various chest diseases and a proper rationale will be necessary to treat several conditions with minimal side effects.

IN ASTHMA

In status asthmaticus the best preparation is Hydrocortisone hemisuccinate and is given in high doses e. g. 1000 mgm a day. A very high dose of 4 mgm/kg per 140 mts in first 24 hrs. I. V. has also been advocated. Advantages are that it is water soluble and can be given as a "Slug-Intravenous". I. V. Steroids are required for first 24-48 hrs. after which oral teratment is given.

Amongst oral steroids, choice, is Prednisolone, due to its low sodium retaining effect. Dose is 30 mgm daily in divided doses with reduction of 5 mgm per day, in one week. Maintenance therapy can be given with Prednisolone not more than 10 mg. a day.

Side effects of Corticosteroids appear when more than 10 mgm of Prednisolone or salts are given. Features are:-(1) Cushing'es syndrom (2) Suppression of endogenous steriod production (3) Retarded growth in children (4) Withdrawal symptoms.

Prevention of side effects is by (1)giving drugs only 3 or 4 days in a week (2)giving drugs on alternate days once relief is obtained (3) use of ACTH including new preparations like tetracosactrin derivatives and synacthen depot (4) use of Disodium cromoglycate and reduction of steroid dosage (5) steroid aerosol using Beclomethasone diproprionate (6) increasing steroid dose during stress and operations, and (7) By confirming that patient has not been taking steroids before operation is contemplated.

OTHER DISEASES—Steroids are also useful in—

1. Severe cases of pulmonary sarcoidosis, cryptogenic fibrosing alveolitis of recent origin and some cases of polyarteritis nodosa presenting as asthma.
2. In tuberculosis, effectiveness of steroids has narrowed down to pleural effusion and severely ill patients of miliary tuberculosis with or without TB meningitis. On the other hand steroids may cause reactivation of latent focus.

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ABSTRACT

PULMONARY THROMBOEMBOLISM PRESENTING AS ASTHMA

Winderbank W. J. et. al;

Brit. Med. J. Vol. 1 pp 90-94, Jan 1973.

"Embolic-induced Asthma" was first discovered during life by Gurenich et al. Bronchospasm has been demonstrated following experimental thrombolism in dogs. Broncho-constriction was found in 85% of patients who had suffered from recent pulmonary emboli by Sasahara in 1967. The present workers found 12 cases out of 250 proved cases of pulmonary embolism to have bronchoconstriction manifested by wheezing and presenting with initial diagnosis of Bronchial Asthma. Age of these patients varied from 39-64 years relatively late in life. The duration of "asthma" ranged from 2-10 years. Only two had the 'attack' for the first time. Only 2 had a family history of asthma, though 6 out of 12 had an allergic diathesis, in the form of sputum or blood eosinophilia.

PaO_2 and $PaCO_2$ were measured in 6 patients–2 had PaO_2 reduced below 50 mm-Hg, and four had 62-64 mm Hg, $PaCO_2$ was raised above 40 mm Hg in 3 cases.

FEV_1 was reduced 51% to 70% of VC in these patients.

Pulmonary Angiography: showed intravascular filling defects, cut-off appearance of vessels and alveolar opacities (Grade I) in 4 patients and Focal decreased flow, distorted vessels, crowding of vessels and attenuation of vessels (Grade II) in 7 patients, thus establishing the diagnosis of pulmonary thrombo-embolism in all these patients who had presented as Bronchial Asthma. This proved the investigation of choice in preference to isotope scanning. There was mean Pulmonary Arterial Pressure which was raised in seven, one of whom had mitral valvular disease.

The pulmonary angiograms were repeated after five days of treatment and showed improvement further proving the diagnosis of Pulmonary embolism.

The cause of bronchial constriction in pulmonary embolism is not established but experimentally release of Serotonia from platelets on the clot has been demonstrated. The release of Serotonin can be prevented by I. V. Heparin, which prevents the onset of bronchoconstriction. In contrast the broncho-constriction related to bronchitis and emphysema has not been relieved by Intravenous Heparin.

The other mechanism for bronchoconstriction may be decreased in $PaCO_2$ in the alveoli distal to the occlusion. This has been shown experimentally in animals.

Half the patients had an allergic diathesis. It has been supposed by the authors that such persons with abnormally reactive bronchi, respond to a substance related to the pulmonary emboli, by bronchoconstriction.

Comments of the Authors : To definitely associate pulmonary embolism in a patient who presents with wheezing resembling bronchial asthma–following may be kept in mind :—

(a) Features of right failure, venous abnormalities in lower level and haemoptysis supported by E.C.G., X-ray abnormalities of pulmonary embolism.

(b) Associated condition e. g. pregnancy, surgical operations, oral contraceptives.

(c) Failure to respond to conventional therapy of bronchial Asthma.

(d) "Bronchial Asthma" starting comparatively late in life.

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ABSTRACT

SMALL AIRWAY DISEASE IN ASYMPTOMATIC ADOLESCENTS

D. J. Hill et. al,

American Review of Respiratory Diseases. 106, 6 Dec 1972, p. 873

To determine the frequency of persistent obstruction of the smll airways in adolescents with asthma during asymptomatic periods, frequency dependence of dynamic compliance was measured in 15 adolescents. The cases were selected from 500 children who had normal respiratory resistence and lung volume. Eight of the fifteen adolescents had frequency dependence of dynamic compliance; its presence did not correlate with the number of episodes of overt wheezing or the time lapse from the most recent episode. Frequency dependence of compliance in 5 of the 7 cases was abolished after an inhalation of isoproteranol. The study indicated that small airway obstruction persisted in some adolescent during asymptomatic periods but its presence could not be predicted on the basis of clinical findings. To determine its significance will require a similar method of detection than measurement of frequency dependence of dynamic compliance.

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ABSTRACT

HEMODYNAMIC EFFECTS OF MORPHINE AND PENTAZOCINE DIFFER IN CARDIAC PATIENTS

Edwin, L., Alderman, M. D., William H. Barry, M. D.,
Anthony F. Graham, M. D., and Donald, C. Harrison, M. D.

New Eng. J. of Med. Vol. 287, 28 Sep 1972, No. 13.

The hemodynamic effects of 8 mg of morphine given intravenously to eight patients were compared in a randomized, double-blind protocol with the effects of 48 mg of intravenous pentazocine given to nine patients. All 17 patients had arteriographically proved coronary-artery disease. Administration of morphine significantly decreased oxygen consumption (-11.6 per cent), cardiac index (-8.6 per cent) and left ventricular end-diastolic pressure (-20.5 per cent). In contrast, pentazocine significantly elevated mean aortic pressure (+12.5 per cent), left ventricular end-diastolic pressure (+20.2 per cent) and mean pulmonary-artery pressure (+36. 3 percent). Because of the different effects of morphine and pentazocine on cardiac output and arterial pressure, cardiac work was decreased 8.2 per cent by pentazocine. The differing hemodynamic effects of morphine and pentazocine suggest that consideration should be given to each patient's cardiovascular status before administration of these analgesic drugs and to the hemodynamic effects of the agent selected.

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ABSTRACT

THE EFFECTS OF PHENOBARBITAL ON BILE SALTS AND BILIRUBIN IN PATIENTS WITH INTRAHEPATIC AND EXTRAHEPATIC CHOLESTASIS

Adolf Stiehl, M.D., M. Michaeal Thaler, M.D. and William H. Admirand, M.D.

The New Eng. J. of Med. 20 Apr 1972 p 858

In two children with intrahepatic cholestasis treated with phenobarbital (10 mg per kilogram of body weight per day) for four days, serum bile salt concentration decreased from 100 to 400 to 1 to 10 ug per milliliter, the pruritus disappeared. The serum bilirubin concentrations were reduced to 20 to 50 per cent of pretreatment values, and the ^{131}I-Rose Bengal fecal excretion increased during treatment. In contrast, phenobarbital had no effect on serum bile salts, bilirubin, ^{131}I-Rose Benga l excretion and pruritus in a child with extrahepatic biliary obstruction. Decreased serum bile salt concentrations and concomitantly increased fecal excretion of Rose Bengal in phenobarbital-treated patients suggest that the barbiturate stimulates bile secretion and biliary excretion of bile salts. It may be helpful in management of young patients with intrahepatic cholestasis.

Prephared by : Brig S. N. Basu,
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PUNE-1.

ABSTRACT

GALL STONES AND CHOLESTEROL-LOWERING DIETS

Struvendent et al

New Eng. J. Med. 288, 1, 24, 1973

To study the effect of long term feeding of a high unsaturated fat diet, also low in saturated fat and cholesterol on the incidence of gall stones in man, autopsy records of the Los Angeles Veterans Administration were reviewed; the cases belonged to the group understudy on long term basis to assess effects of diet on aterogenesis.

It was found that those who had more than 33% of the experimental meals during life were more likely to have gall-stones (34%) than controls (14%). Gall stone formation correlated well with (a) number of experimental meals eaten, and (b) over weight to the extent of 9.1 Kg. or more.

This study highlights the probability that diets containing high ratio of unsaturated to saturated fats, high ratio of plant sterol to cholestrol, or both, are likely to cause gall stones.

Prepared by: Group Capt S. Krishnamurti,
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PUNE-1

ABSTRACT

ALLERGIC INTERSTITIAL NEPHRITIS

Lyons et al

New Eng. J. Med. 288, 3, 124, 1973

Varying degree of renal failure develops in cases of nephrotic syndrome. If such renal failure progresses faster than indicated by the natural history of the etiology of the nephrotic syndrome, allergic interstitial nephritis should also be considered besides other causes of acceleration renal failure.

Eosinophilia in blood of a skin rash may call attention to the allergic aetiology but neither is invariable. In all cases renal biopsy is the only diagnostic and it shows diffuse infilteration of the interstitium with eosinophils.

In the 4 cases reported, frusemide/thiazides were stopped and steroids were given and dramatic improvement was noted. As they were unable to prove any particular allergen, the authors speculate that the interstitial nephritis in their cases may be due to the diuretics used.

Prepared by : Group Capt S. Krishnamurti,
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ABSTRACT

PULSUS PARADOXUS AND AIRWAYS OBSTRUCTION

Rebuch & Pengelly

New Eng. J. Med. 288, 2, 66, 1973

Pulsus paradoxus was found in 34 out of 76 cases of Asthma; this phenomenon was noted in those patients who had greater degree airways obstruction.

The authors conducted a study on 5 normal subjects, simulating the various mechanical changes in cases with pulsus paradoxus. They found a combination of increased FRC (54-78% rise above resting level) and high intra-alveolar pressure (14-22 cm of water) produced pulsus paradoxus; neither factor alone was associated with its occurrance.

The authors conclude from these findings that the mechanism responsible for pulsus paradoxus in severe asthma is the combination of cardiac compression, due to over inflation of lungs and the high intra-pulmonary pressure, due to airways obstruction.

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PUNE-1.

ABSTRACT

GIVING DRUGS BEFORE OR AFTER FOOD

Drugs & Therapeutics Bulletin. Vol. 10, No. 3. 4 Feb 1972.

1. The timing of drug administration in relation to food can have an important influence on the effects of the drug.

2. Presence of food in the stomach may reduce or slow the absorption of some drugs. Certain foods can reduce the total absorption of some drugs e.g. food rich in calcium, magnesium or iron, insoluble complexes are formed with tetracyclines thus reducing their absorption.

3. Following antibiotics should be taken half an hour or more before food.

    Erythromycin base lincomycin

    Oral penicillins
    (ampicillin,
    cloxacillin,
    flucloxacillin,
    penicillin-V
    phenathicillin)

    Rifampicin
    Tetracycline

4. Dietary constituents may enhance absorption of some drugs e. g. Griseofulvin is absorbed better with fatty meal.

5. **When to give certain drugs in relation to meals**

Antihelminthics

Bephenium (For round and hook worms) Dichlorophen & niclosamide (for tape worms)	on an empty stomach to improve contact with worms.

Appetite suppressants

Fenfluramine; diethyl propion	Well before meals for optimum psychological and pharmacological effectiveness

Sulphonyl ureas Tolbutamide	—with or just before meals for optimum control of blood sugar,
glibenclamide	—after the first main meal to prevent hypoglycaemia
Pancreatine Preparation	—immediately before or with food
Metoclopramide	—Just before meals for patients suffering from flatulent dyspepsia immediately after eating; with or after meals where these symptoms arise an hour or more after the meal.
Mucosal protectant Polymethyl siloxane	—before meals

6. Some of the drugs which are likely to cause gastric symptoms when taken on empty stomach are listed as under :—

- cyproheptadine
- diphenhydramine
- ethacrymic acid
- ethionamide
- indomethacin
- most iron preparations
- levodopa
- mepyramine
- nitrofurantoin
- oxyphenbutazone
- phenylbutazone
- promethazine
- salicylates
- sodium fusidate
- triprolidine

Prepared by : Group Capt S Krishnamurti,
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PUNE—1.

ABSTRACT

LONG TERM LOW-DOSE CHEMOTHERAPY FOR RECURRENT URINARY TRACT INFECTION

Drug and Therapeutic Bulletin; Vol. 10, No. 11, 26 May, 1972.

Treatment of urinary tract infection aims at relieving distressing symptoms and to prevent scarring with subsequent deterioration in renal function.

Clinical trials of sulphonamides, hexamine mandalate and nitrofurantoin in the ordinary dosage have demonstrated their ability to prevent recurrences of bacteriuria. Success seems unrelated to the presence of radiological abnormalities.

Low-dosage regimens : Sulphadimidine lG, nitrofurantoin 100 mg or cycloserine 125 mg given as single evening doses were compared over 6 months in 81 women with E. coli infections. The results were similar for all three treatments. The over all recurrence rate was 16% during treatment and it rose upto 36% during the six months after therapy had been stopped.

In another trial nitrofurantoin 100 mg, hexamandalate 1G (without acidification) or placebo was given nightly to 45 women with kidney or bladder infections. Only nitrofurantoin - significantly reduced bacteriuria, pyuria and the incidence of symptoms in both types of infection. However, hexamine did reduce bacteriuria and pyuria when the bladder was infected. In a recent carefully controlled double blind trial an even smaller dose of nitrofurantoin (50 mg/night) was much better than placebo in preventing relapse of bacteriuria in 50 women without radiological abnormality.

In an uncontrolled series of 52 patients cortimoxazole (septrin) taken at night effectively suppressed bacteriuria. Some patients required a tablet only twice weekly.

Unwanted Effects

Gastro intestinal complaints may be reduced by lowering the dose of nitrofurantoin to 50 mg/day.

With impaired renal functions increased toxicity must be anticipated.

Hypersensitivity reactions are prominent among the adverse effects of sulphonomides but serious ones are rare.

Cycloserine may cause mental disturbance but the true incidence of this effcet is not known.

Emergence of resistant organisms with any of these drugs so far has not been a major problem.

Choice of Drug

One daily bed-time dose of either nitrofurantoin 50 mg or sulphadimidine 1G has most to offer in preventing recurrent infection even in patients with radiological abnormalities. Serious unwanted effects are uncommon.

Daily dose is best taken at night so that high concentrations of drug are obtained in the urine when flow rates are lowest.

To minimise stasis, the patient should drink at least 3 litre/day of water.

Prepared by : Group Capt S. Krishnamurti,
Offg. Professor of Medicine,
Armed Forces Medical College,
PUNE-1.

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STUDIES ON THE AEROBIC WORK CAPACITY IN GORKHAS - CIVILIANS AND SOLDIERS

BY

Lt Col S. K. Das* AMC

The Gorkhas are well known for their physical endurance and so also the Gorkha soldiers in our army. It is known that physical training improves the aerobic work capacity of a man by improving his ability to meet the demand for oxygen during severe work. The Gorkha as well as other soldiers undergo a regular systematic physical training and as a result, are expected to have a high standard of aerobic work capacity. This was already demonstrated in a previous study (Das — 1968). Similar studies in Gorkha civilians are not known. Hence an attempt was made to study the metabolic rate of Gorkha civilians at submaximal and maximal work rate in order to compare their metabolic data with those of the Gorkha soldiers. Such a study is expected to throw some light regarding the effects of physical military training on the aerobic work capacity of the soldiers in general.

MATERIALS AND METHODS

Ten Gorkha civilian who volunteered for this work were drawn from a local source in Calcutta. They had come from Nepal and were staying in Calcutta during the last 2-5 years. By occupation they were engaged as gate keepers or domestic workers and thus engaged in jobs requiring light or moderate manual labour. They were not engaged in vigorous regular physical activities or athletics of endurance type. However, they used to take part occasionally in outdoor activities involving certain amount of muscular exercise. The physical characteristic of the Gorkha civilians is given in Table I.

The description of the Gorkha soldier is also given in Table I. The experimental details are given below.

Maximal oxygen uptake was measured during treadmill run uphill. The speed was kept constant and the load being increased by increasing the gradient of the treadmill.

---

* Physiologist INHS ASVINI, Colaba, Bombay 5.

Two submaximal work loads were used for the Gorkha civilians. The subjects ran for 5—6 minutes at the submaximal loads, respiratory and circulatory measurements being taken during the last minute of the exercise period. The subjects attempted to reach their maximal level by performing one or two exhaustive runs and the respiratory and circulatory measurements being taken during the last minute.

The expired air were collected in Douglas bag and analysed by means of Scholander's 0.5 ml gas analyser (1947). Expired air volumes were measured using Max planck Gashur (Kofranyi and Mitchelis, 1940).

Heart-rates were measured from the electrocardiogram taken during the work.

Linear relationship between oxygen uptake and work load at maximal rates was established and the work level found at which a further increase in work output did not bring about an increase of oxygen up take. The levelling off of oxygen uptake was used as a criterian that the maximal value was reached. Fig I would demonstrate how this criterian was achieved for the soldiers as well as civilians. The civilians could not work beyond 1800 Kilopond meter minute except one subject whose oxygen uptake was also levelled off at higher work loads.

RESULTS

The mean values of pulmonary ventilation, oxygen uptake, heart rates, oxygen extraction factors and RQ along with the results of statistical analysis of the data in respect of the Gorkha civilians were given in Table II and similar data in respect of Gorkha soldiers were represented in Table III.

The mean values of pulmonary ventilation, oxygen uptake, heart rate of the two groups were plotted against various work load as shown in Fig I (a), I (b) and I (c).

Further the mean values of oxygen uptake at various work rates were plotted against Heart rate for the two groups as shown in Graph II. It was seen that the soldiers maintained a linear relationship between heart rate and oxygen uptake with increasing work rate upto the maximal nivea but not the civilians, for them it was curved.

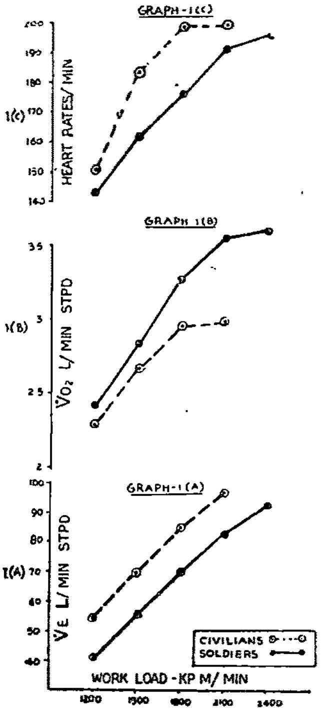

Fig 1 (a-c)-The mean values of pulmonary ventilation litres/min oxygen uptake values litres/min, Heart rate beats/min in relation to work load.

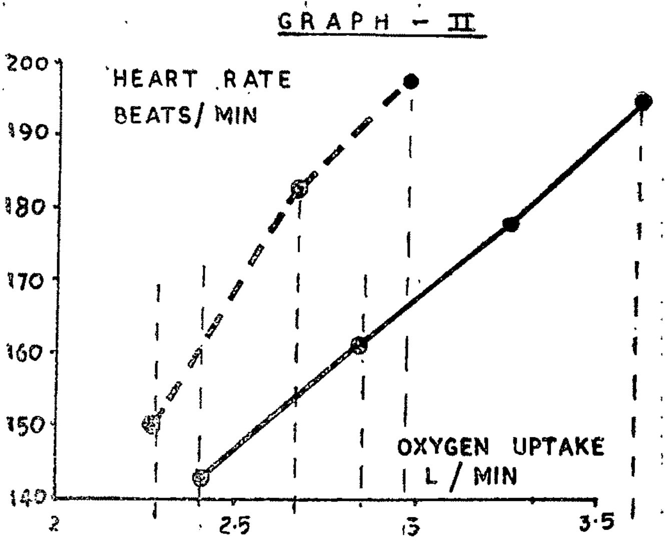

Fig. 2—The mean values of oxygen uptake litres/min in relation to Heart rate beates/min.

Further the mean values of oxygen uptake at different work rates were plotted against ventilation volume for both the groups as in Graph III. It was observed that the ventilation volume at fixed rate of submaximal work were more in civilians.

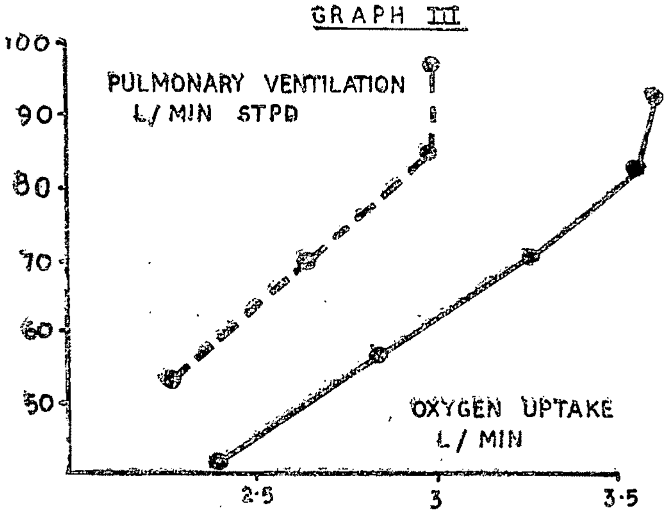

Fig. III—The mean values of oxygen uptake litres/min in relation to ventilation volume litres/min.

Table I

Description of the subjects

	Age Years.	Height cms	weight kg	BSA Sqm
Gorkha Civilians	23.10±1.72 (5.43)	165.50±1.39 (4.39)	61.10±1.20 (3.81)	1.68±.02 (.07)
Gorkha Soldiers	25.32±.38 (3.08)	162.46±.56 (4.54)	59.95±.46 (3.70)	1.64±.01 (.08)

Values are mean ± SE (Standard error of the mean)
Number in parenthesis - SD (Standard Deviation)
BSA = Body Surface Area in Square meters.

Table II

Metabolic data - Gorkha Civilians

Work Rate @ kpm/min	Heart Rates Beats/min	Pulm Ventilation L/min	Oxygen Uptake L/min	Oxygen Uptake ml/kg/min	Oxygen Extraction Factor ml/100 ml air	RQ	Mvv(Mean) L/min (Btps)
1200	150.20 2.58 (8.17)	53.90 1.72 (5.45)	2269.30 79.28 (250.72)	37.20±1.22 (3.87)	4.22±.12 (38)	0.89±02 (.05)	
1500	183.40 ±1.65 (5.22)	69.20 ±1.80 (5.65)	2635.20 ±78.15 (247.13)	43.80±1.05 (3.31)	3.83±.14 (.43)	1.04±.02 (.06)	129.80±4.13 (13.07)
1800	198.60 /±1.68	83.60 ±2.26 (7.14)	2974.90 ±102.68 (324.71)	48.70±1.77 (5.60)	3.58±1.5 (.49)	1.07±.03 (.08)	
*2100	200	96	2935	—	—	—	

*One subject only.

Figures indicate mean values ± standard error of the mean (SE of Mean) Figures in parenthesis indicate standard deviation (SD)

@ Kilopond is unit of force acting on 1 kg at normal acceleration of gravity.
The work unit is Kilopond meter minute (kpm/min).
BTPS —Body Temperature, ambient Pressure and Saturation.
MVV —Maximum Ventilation Volume.

TABLE III

Mean Metabolic Responses to Various work load

Work Loads Kpm/min.	Ventilation L/min Stpd	Oxygen Extraction M1/100 ml air	RQ	Oxygen Uptake ml/min Stpd	Oxygen Uptake ml/kg/min Stpd	Heart Rates per min	MVV (Mean) L/min (Btps)
1200	40.58±.39 (3.16) CV – 7.79	5.95± .03 (.21) CV – 3.53	0.86±.04 (.31) CV – 36.05	2413±17.23 (138.87) CV – 5.75	40.26±.25 (2.02) CV – 5.02	143.17±1.09 (8.85)	
1500	55.71±.59 (4.75) CV – 8.53	5.10±.004 (.04) CV – .78	.93±.02 (0.19) CV – 20.43	2835.49±25.78 (207.75) dv – 7.33	47.38±.28 (2.28) dv – 4.81	162.17±0.92 (7.38)	
1800	69.72±.91 (7.37) CV – 10.57	4.72±.04 (.36) CV – 7.63	1.01±.01 (.06) CV – 5.94	3279.08±41.13	54.55±.48 (3.09) dv – 7.15	177.71±0.87 (7.01)	140.63 ±2.23
2100	83.83±1.21 (9.78) CV – 11.67	4.26±.05 (.44) CV – 10.33	1.06±.01 (.06) CV – 5.66	3552.65±49.46 (397.79) CV – 11.20	59.17±.58 (4.69) CV – 7.93	192.00±0.80 (6.42)	(17.84) CV – 12.7
2400	91.57±1.45 (11.67) CV – 12.7	3.96±.06 (.48)	1.10±.01 (.09)	3611.55±47.95 (386.59) CV – 10.7	60.20±.58 (4.64) CV – 7.7	196.03±.94 (7.59) CV – 3.8	

Values are mean ± SE (Standard error of the mean)

SD – Numbers in parenthesis (Standard deviation)

CV – Coefficient of variation. STPD – Standard temperature, pressure and dry air.

Kilopond is unit of force acting on 1 kg at normal acceleration of gravity.

The work unit is kilopond meter minute (kpm/min).

DISCUSSION

Submaximal work rate :- It was observed that the oxygen uptake values during treadmill run submaximally at a fixed rate were somewhat higher in soldiers than in the civlian subjects. The reason for this was not known. Astrand (1960), Hermensen (1965) also made similar observations for athletes in comparison to non-athletes. Moreover at lower submaximal work rates no significant defferences in metabolic rates in respect of athletes and non-athletes were noticed by the said workers. Maximal work rate: - It was clearly demonstrated that working capacity of the soldiers was better than that of civilians. The maximal limit of work for soldiers was at 2400 kpm/min whereas the same for the civilians was at 1800 kpm/min except one subject who worked upto 2100 kpm/min.

Maximal oxygen uptake :— The maximal oxygen uptake values (mean) for the soldiers were 3.6 litres/min or 60.2 ml/kg/min and that for the civilians were 2.97 litres/min or 48.64 ml kg min. The maximal oxygen uptake values for the soldier were 21.4% when expressed in terms of per kg of body weight more than that of the civilians. So it was obsereved that the soldiers had greater maximal oxygen uptake values than the civilians. Therefore, the soldiers superiority in carrying oxygen from the ambient air to the working muscles was clearly demonstrated.

According to Saltin and Astrand (1967), Ekblom and Hermansen (1968), the Static and functional dimensions of the athletes were much more developed than that of the non-athletes. It means that their maximal cardiac output and maximal stroke volumes as well as heart volumes were relatively large and well related to their maximal orygen uptake. During severe exercise the athletes oxygen transport mechanism (i.e. extraction of oxygen from the ambient air, transportation of O_2 by cardiovascular system and utilisation of oxygen by working muscles) was very effective (Ekblom 1969).

The highest individual values of maximum oxygen uptake of the top athletes as noted by different workers - (1) Robinson et al (1937) 5.35 litres/min. (2) Saltin and Astrand (1967) 85.1 ml/kg/min. (3) Ekblom & Hermansen (1968) 81 ml/kg/min. (4) Das (1968) 71 ml/kg/min and the average values of oxygen uptake for the athletes - (1) Astrand (1952) 66.8 ml/kg/min (2) Landy's (cited by Wyndham et al 1959) 76.6 ml/kg/min, (3) Hermansen & Andersen (1965) 71.0 ml/kg/min and (4) Das (1968) 60.20 ml/kg/min.

The average values for maximal oxygen uptake of sedentary people as noted by various workers - (1) Robinson et al (1937) 49.9 ml/kg/min. (2) Taylor et al (1955) 48.3 ml/kg/min. (3) Wyndham (1963) 48 ml/kg/min. (4) Hermansen (1965) 44 ml/kg/min. and Das (earlier work) 41 ml kg/min.

These reported values of maximal oxygen uptake in respect of sedentary men would compare favourably with the figures of maximal oxygen uptake of the civilians (48.70 ml/kg/min) as found in the present study.

Heart rate : Studying the heart rate at submaximal work rates of the two groups, it was observed that the increment in heart rate for a given increment in metabolic rate was smaller in soldiers than in civilians (Fig II and Fig I (c) and it was more marked at a work rate demanding 60-90% of maximal oxygen uptake of the subjects. Hermensen (1965) observed that the elevation of metabolic rate did not bring about the heart rate increase in athletes in as sedentary subjects, well trained young men would increase either the stroke volume or A.V. difference or both more than untrained men when the load of exercise was increased progressively from light to heavy.

Ventilation :- It was observed in this study that the elevation of metabolic rate brought about more increase of ventilation volume in case of the civilians than the soldiers (Fig III and 1 (a).

The well known exercise induced hyperventilation started at a metabolic level of about 50 to 70% of maximal aerobic capacity both in soldiers and civilians. This was in agreement with Astrand (1960) and Hermensen (1965) who observed the hyperventilation in their subjects started out of proportion at 70-80% of their metabolic level. The pulmonary ventilation efficiency was better in soldiers than in civilians.

RQ and Oxygen Extraction Factor values :- The values of RQ were comparatively higher and the oxygen extraction values were lower in civilians than that of the soliders at almost all work rates investigated.

SUMMARY

This paper deals with the problem of assessing the work capacity of a group of Gorkha civilians and to compare it with that of Gorkha Soldiers. The maximal oxygen uptake (Vo^2 max), the ventilation rate (VE), the heart rate (HR) beats/min for the civilians were 2936 ml/min or 48.64 ml kg/min, 98 litres/min and 198 beats/min respectively and the corresponding figures for soldiers were 3862 ml/min or 60.20 ml kg min 96 litres/min and 196 beats/min. It was noted that the soldiers maintained a linear relationship at a fixed submaximal and maximal work rates between oxygen uptake and heart rate values whereas the civilians did not, linear relationship became curved.

Oxygen cost of treadmill run at submaximal work rates of the soldiers were higher than that of the civilians whereas the civilians had higher values of ventilation volume and heart rate at the said work rates.

The civilians also had lower MVV values. The oxygen uptake values at maximal work rate were lower than MVV values for both the groups.

The V$_{62}$ max values of the soldiers were 21.4% (in terms of body weight) higher than that of the civilians. The physical characteristics as well as gene of the civilians were similar to that of the soldiers. Hence the higher aerobic capacity of the soldiers could only be attributed to physical training background of the soldiers.

CONCLUSION

From the result, it was observed that in comparison with the civilians the soldiers at all work rates had :—

(a) Higher physical work capacity.
(b) Higher oxygen uptake values.
(c) Lower heart rate (At maximal work there is hardly any difference of heart rates in between the two groups).
(d) Lower RQ values.
(e) Lower pulmonary ventilation volume.
(f) Higher oxygen extraction values.

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AN EPIDEMIOLOGICAL STUDY OF INJURIES IN ARMY PERSONNEL AT A PEACE STATION

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INTRODUCTION

Deaths due to infections and nutritional disturbances have fallen to a level considered unattainable only a few years ago. On the other hand the death rates due to accidents remain high and for many types of accidents have increased. It is not only as a cause of death that accidents are important but the large number of non fatal injuries, they cause, between 100-200 times greater and sometimes even more (Mc Farland, 1963). This gives some indication of the sufferings and loss, and sometimes permanent disablement and disfigurement, that is involved. Quite apart from the problem of medical care, social policy and economics involved, accidental injuries like all serious threats to health have human repercussions, that are difficult to measure; grief, separation, pain, family disruption, chronic handicap and deformity. Almost nothing is known of this human cost except, perhaps certain types of accidental injuries may have more serious repercussion (W. H. O. Chronicle, 1966).

The present investi gation was an attempt to study the distribution and determinants of accidental injuries in armed forces personnel, their social and economic impact in order to formulate research programmes and preventive measures for all types of injuries.

MATERIAL AND METHODS

The study was carried out at the cantonment areas of Allahabad from September 1966 to June 1967. The universe of study comprised of all troops stationed at Allahabad Cantonment totalling nearly 5,670 with percentage of Officers, J.C.O.'s and other ranks being nearly identical with that of the Indian Army in general. The samples were selected from all cases of injuries admitted to Military Hospital, Allahabad and all cases of injuries treated in various medical inpection rooms in cantonment areas. The controls were taken from those who were admitted to Military Hospital or were treated in different M.I. rooms for the ailments other than injuries. The study was prospective in nature as the sequence of events were recorded in a given period of time. Collection of information for every single injury was done on a prepared schedule by personal inter. view during the period of treatment and convalescence information about controls was recorded on a different schedule.

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RESULTS

The number of injury cases during the study period was 253, the incidence rate of the combined admitted and non admitted cases being 54.5 per 1000. The incidence rate of admitted cases only was 27. 1 per 1000 and injuries accounted for 9. 86 per cent of total admissions. The rate of average daily sick in hospital for all diseases was 16.55 per 1000 and for injuries it was 2.31 per 1000. Daily manpower lost due to injuries was 4.54 per 1000. Mortality rate for injuries was 0.43 per 1000.

Table I

Distribution of injured and controls by age groups

Age (in years)	Injured Number	Injured Percentage	Controls Number	Controls Percentage
15 - 19	7	2.76	3	1.18
20 - 24	124	49.01	82	32.41
25 - 29	61	24.11	91	35 96
30 - 34	30	11.85	36	14.22
35 - 39	17	6.71	19	7.51
40 - 44	11	4.34	14	5 53
45 - 49	3	1.18	8	3.16
Total	253	100.00	253	100.00

It is seen from table I that the largest number of injuries (49.01%) occurred in the age group 20-24 years Whereas, age group 25–29 years contributed maximum number of admission (35 96%) for Sickness (Table 1). The association was significant of age with injuries (x^2=18. 10 df. -4, $P<0.01$).

The injuries occurring to married persons were 150, while 163 controls belonged to this group as compared to 96 and 80 respectively among unmarried persons. Those with length of service between 1-4 years had 96 (36.75%) injuries and in 5–9 years group 87 (34.38%), in controls these groups showed 72 (28.38%) and 91 (35.96%) respectively of total admissions other than injuries.

Table 2

Distribution of injured and controls by trades.

Trade	Injured		Controls	
	Number	Percentage	Number	Percentage
General duty	62	24.50	98	38.73
Drivers	38	15.02	20	7.90
Clerks/Storeman	38	15.02	40	15.91
Vehicle mechanic	22	8.69	11	4.34
Artificier	23	9.09	23	9.09
Engine fitter	9	3.55	7	2.76
Craftman	9	3.55	10	3.95
Driver plant	3	1.18	8	3.16
Machinist	6	2.37	0	0.00
Electrician	2	0.78	8	3.16
Wireless operator	6	2.37	1	0.39
Signalman	5	1.97	1	0.39
Cook	3	1.18	6	2.37
Driver animal transport	4	1.58	2	0.78
Others	23	9.09	18	7.11
Total	253	100.00	253	100.00

In the tradewise break up largest number of injuries (24.50%) occurred in general duty personnel. In controls to the same category of personnel had higher incidence of diseases being 38.73 per cent (Table 2). The association of trade with injuries was significant ($X^2 = 14.82$, df - 6, $P<0.02$).

Table 3

Distribution of injured and controls by literacy status.

Literacy status	Injured		Controls	
	Number	Percentage	Number	Percentage
Illiterate	12	4.74	8	3.16
III Class English	62	24.50	114	45 06
II Class English	91	35.97	59	23.32
I Class English	88	34.73	72	28.45
Total	253	100.00	253	100.00

Largest number of injuries (35.97%) occurred to soldiers who passed army II class English standard test of the total. In controls it was more in those having passed

III class English test being 45,06 per cent (Table 3). The association of literacy status with injuries was significant ($X^2 = 24.88$, df - 3, $P<0.01$).

Most of the injuries had occurred in daylight hours i e., 12.00 hours to 18.00 hours number was 108 (42.69%) next being, between 0800 hours to 1200 hours which was 108 (42.69%) minimum injuries occurred between 1800 hours to 0600 hours being 88 (11.06%).

As far as the place of occurrance is concerned, maximum number of injuries 70 (27.66%) occurred in unitlines, while in training ground and play ground 57 (22.53%) injuries occurred in each place. Next being place of work 43 (16.99), highways, Railway station and village homes accounted for 13 (5.02%). 10 (3.95%) and 3 (1.18%) respectively.

Maximum injuries (41.89%) occurred due to falls followed by hit against or by an object (37.54%). Automobile accidents (3.95%) and machine accidents (3.16%) were other common types (Table 4).

Table 4

Distribution of injuries by types.

Type of injury	Number	Percentage
Falls	105	41.89
Hit by or against an object	95	37.54
Automobile	10	3.95
Sudden twisting of leg	10	3.95
Machinary accident	8	3.16
Homicide	6	2.37
Caught in a device	4	1.58
Self inflicted injury	3	1.18
Thermal injury	2	0.78
Glass injury	2	0.78
Others (Accidents - Rail, Rickshaw, Fire and roof fall 1 each)	4	1.58
Total	253	100.00

As a result of these accidents four common nature of injuries occurred which were contusion 104 cases (41.10%) lacerations 52 (20.59%), sprains/strains 47 (18.57%) and fractures 33 (13.04%). These were followed by head injuries 4 (1.58%), dislocation 3 (1.18%), burns 3 (1.08) and others like synovitis etc. accounted for 7 cases (2.76%). In the nature of severity, 198 (78.26%) were mild, 19 (7.51%) moderately severe, 34 (13.43%) were severe while 2 (0.78%) proved fatal.

One hundred and twenty nine cases which were admitted to the hospital accounted for 1,854 days of hospetal stay. Average duration of stay in hospital per patient of injury was 14.37 days as compared to 9.2 days per patient of sickness (Table 5).

Table 5

Distribution of hospital stay by rank

Rank	Duration of hospital stay (days)			
	Injured		Controls	
	Total	Per case	Total	Per case
Officer	162	20.25	4	4.00
JCO	19	9.50	50	8.33
NCO	312	13.56	330	9.16
OR	1141	12.96	741	9.50
Recruit	28	5.60	32	6.40
NCE	192	64.00	40	13.33
Total	1854	14.37	1197	9·20

In all 595 days were spent in convalascence by all cases of injuries without any output of work, in addition 32 days spent in light duties, while 168 days were spent in convalascence by all cases of diseases without any output of work.

Table 6

Distribution of man d ys wastage by rank.

Rank	Man days wastage			
	Injured		Controls	
	Total	Per case	Total	Per case
Officers	162	20.20	4	4.00
JCO	53	17.60	78	6.00
NCO	655	10.90	577	8.20
OR	1842	10.90	1098	6.30
Recruit	81	10.10	32	6.40
NCE	195	46.70	40	13.33
Total	2988	11.81	1829	7.20

The man days wastage (2,988) excludes four cases who were transferred to other hospitals. Man days lost per soldier due to injuries (combined admitted and outdoor treated cases) was 0.53 days as compared to 5.95 man days lost per soldier due to all diseases. Man days lost per soldier due to injuries admitted to hospital was 0.4. Average man days wastage per case of injury was 11.81 days and that of diseases 7.20 days (Table 6). The man days wastage per case of NCEs was abnormally high due to long retention of a patient of railway accident who had traumatic amputation of right leg.

Table 7

Economic loss due to pay wastage of injured by ranks.

Ranks	Average pay per day (Rs.)	Man days lost	Total pay wastage (Rs.)
Officer	25.00	162	4050.00
JCO	7 33	53	388.49
NCO	4.33	655	2836.15
OR	4.03	1923	7749.69
NCsE	2.93	195	571.35
Total	7.27	2988	15,595.63

Note : In order to calculate the economic loss, the loss due to pay wastage only has been considered while other expeneses could not be worked out.

The loss of pay of 253 personnel due to 2,988 man days lost comes to rupees 15,595.63 (Table 7) which means rupees 18,714.00 are lost per year in Allahabad in pay only due to injruies. Assuming that average cost of hospitalisation care is rupees 30.00 per bed per day then the cost of hospitalisation of all injury cases works out to be nearly 55,620.00.

DISCUSSION

The high incidence of accidental injuries in the army at a peace station is an indication that it is a health problem of considerable magnitude. The incidence rate of all injuries was 54.5 per1,000 per year, the rate was high as compared to the Indian army which was 26.69 per 1000 (Army Stat. Org., 1966) which is based on the hospital admission only. It is low as compared to agricultural population of Punjab Villages at 115.6 per 1,000 (Gordon et al, 1962), Central Government Health Services beneficieries at 183 per 1,000 (Seal, 1965) and Civil Population at 275 per 1,000 (C.B.H.I., 1966). The probable reasons for these differences are (a) the strict enforcement of discipline (b) Compulsory observance of preventive measures and (c) Court of enquiry held in almost all cases to ascertain the cause, to fix responsibility etc.

The fatality rate in this study was 0.43 per 1,000 as compared to Indian Army's 0.38 for 1963 (Army Stat. Org. 1966). The rate compares well with the United States at 0.33 per 1,000 (McFarland 1962), in U. P. at 0.09 per 1000 in 1961 which is low.

Injuries of mild severity were to the extent of 78.28 per cent the average man days wastage was 11.81 days per injury (Table 6). This resulted in considerable pay wastage in that the soldier continued to receive his emoluments without output of work, The pay wastage per injury was rupees 61.64 and inclusive of the cost of hospitalisation the economic loss amounted to rupees 281.48 (approximately) per injury. In addition disability produced, the compensation there of and the damage to equipment etc. all add further to the loss.

Injuries due to falls were commonest (41.89%) followed closely by hits by or against an object (37.54%) as shown in Table 4. The later types of injuries compare well with piercing and cutting object injuries (38%) among agricultural workers of Punjab, while falls were reported less common (9.1%) in them (Gordon et al, 1962). The fear of punishment due to failure to line up in time, the rigid enforcement of orders and the youth, agility and exuberence of age are probably the contributing factors to the increased frequency of falls. Falls constituted 48.7 per cent of all injuries among Central Government employees (Seal, 1965).

Contusions and abrasious were the most common injuries constituting 41.10 per cent and lacerations 20.59 per cent of the total as compared to 32 and 35 percent of the total injuries in the Central Government employees (Seal, 1965). Laceration accounted for 50 percent of all injuries among agricultural workers (Gordon et al, 1962). The differences with the other two findings might be due to correct and complete recording of every injury hence minor injuries are more reported in this study.

Nearly 89.31 per cent of injuries occurred in the unit lines, training ground and place of work, 28 percent of all injuries were in unit lines where maximum miscellaneous work is carried out which exposes them to more of injuries. The work in the village homes and daily work of a soldier in unit lines are almost of same nature. Gordon (1962) found 43.1 per cent injuries occurring in home surroundings. Thus injuries in the home surroundings and unit lines have relatively higher frequencies of injuries.

In the present study, 246 injured suffered temporary disability for 2988 days, one case was downgraded to category CEE $\underset{11311}{\text{SHAPE}}$ (Temporary for 24 weeks), advised light duties and exemption from P.T. and Parades temporarily for six months and one to permanent cat. CEE. Two cases of injuries were fatal. Five injured had traumatic amputations of various parts of the body one had a severed right leg causing permanent disability, four others sufferred crush injury of one of the fingers of the hand or toe in foot resulting in permanent disability of less than 20 per cent in each.

The age of the soldier (Table 1), his trade (Table 2) and the literacy status (Table 3) had significant association with injuries. Almost 50 per cent of the injuries occurred in the age group 20 to 24 years, while 32 per cent diseases affected the same age group of controls. The inherent agility of the comparatively young population, more physical work and immaturity are probably the causes for this high incidence, an industrial physician termed accidents as "an affliction of youth" (McFarland, 1963). Seal (1965) and Gordon et al (1962) have reported higher frequencies of accidents in 15 to 24 years of age group and at high level throughout the productivity years of life in their studies respectively. It was also found that the persons with less length of service (1 to 4 years) suffered more from injuries, needless to mention that youth and inexpérience were responsible for this.

Whether literacy makes a person more prone to injuries is difficult to say as appears from the trend of more injuries occurring in more educated persons in this study (Table 3). This might be because the persons in fighting arms, though less educated are more rough and tough and might be more immune to injuries, however this needs to be investigated more, similarly, about the trades. There are various types of trades in the army services some of them expose the personnel more to injuries than others, these are shown in Table 2.

The phenomenon involved in the occurrance of injuries is similar as that of a disease process i.e. an interaction of agent, host and environment. Pathogenesis of injuries arises in the presence of susceptible host and predisposing factors involving environmental variables including agents capable in these circumstances of producing disease or injuries. The detailed investigations of these factors help to design outline for prevention of accidents and injuries, their minimisation and rehabilitation.

The present study had the advantage that there was total and complete recording of all events (injuries) therefore observer variation was eliminated as the same doctor continued to treat the injured and there was complete follow up of injuries because of strict discipline. Court of enquiry held for almost all cases etc. except for four cases transferred away, there was no loss due to attrition.

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AUDIOMETRIC STUDY IN ARMY RECRUITS

BY

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INTRODUCTION

A good physical standard for armed forces personnel can hardly be emphasized as they have to work in various odd and hard situations. The recruits of Indian Armed Forces are given various physical and medical tests before they are inducted into service.Apart from other medical tests, the ears are examined for any perforation or ailment. The hearing acuity is judged only qualitatively by giving whisper and voice tests. Due to administrative difficulties they are not given complete audiometric test which is quantitative, more efficient and reveals the actual hearing status.

The present audiometric study was carried out to judge the efficacy of whisper and voice tests in eliminating recruits with hearing loss.

METHOD AND MATERIAL

The study was carried out on 113 Army Recruits, age 19–22 years with an average service of 3.2 months to their credit. The audiometric test by air conduction was carried out utilising BEL clinical audiometer calibrated according to I SO specification in an acoustic booth. Where necessary bone conduction was also monitored by applying bone conductor in the mastoid region keeping the ear under test unoccluded and masking the contra ear. It was ascertained that clear 16 hrs had elapsed after any noise exposure before audiometry was done as suggested by (Glorig, 1966). A questionnaire was filled to record relevant information which could throw light on ear ailments, aural assault suffered before joining the service and during service besides injury and other biodata.

RESULTS

Out of 113 recruits tested 102 were found having normal hearing acuity, the rest 11 had defective hearing. The threshold of hearing for normal subjects varied from 10.5 dB to 13 dB between the frequency ranges of 125 to 500 cps as well as between 4000 to 8000 cps. At the remaining frequencies the hearing threshold was still better and varied between 8 to 10 dB (Fig. 1). The average audiogram of those showing defective hearing is presented in Fig. 2. It will be seen that hearing threshold

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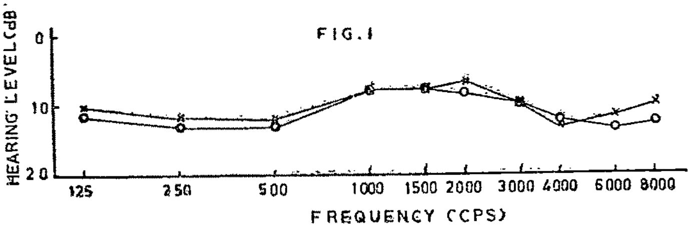

Fig. 1 Hearing level of normal recruits (N=102)

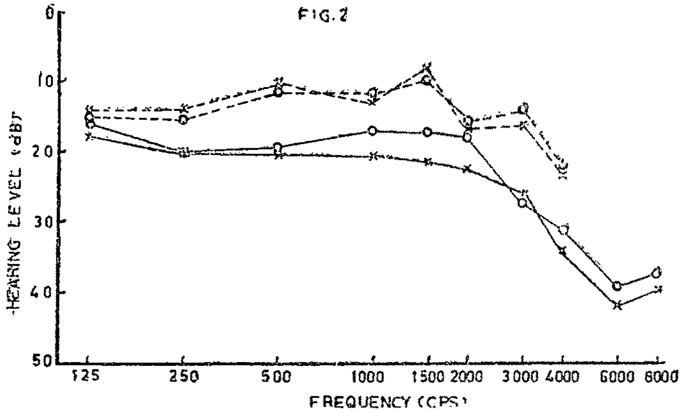

Fig. 2 Audiogram of recruits having impaired hearing (N=11).

in them upto 2000 cps varied from 18 dB to 22.5 dB in the worse ear. But beyond 2000 cps, the hearing loss progressively increased and at 6000 cps it was 41.5 dB with no further fall at 8000 cps. The bone conduction curves also showed depression at higher frequencies and at 4000 cps the bone conduction threshold was 23 dB.

Out of these 11 subjects 3 as well had speech hearing loss of 38, 46 and 50 dB respectively (average loss at 500, 1000 and 2000 cps). Their audiograms for the worse ear are presented in Fig. 3.

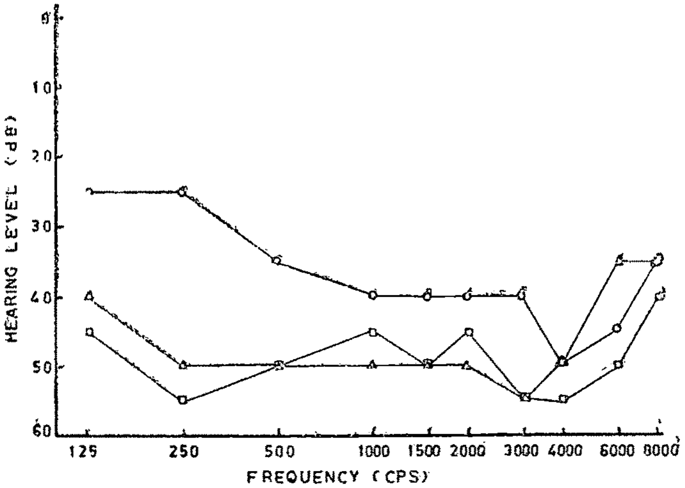

Fig. 3 Hearing level in the worse ear of three subjects.

DISCUSSION

Eleven out of 113 subjects were found to have hearing loss forming about 10% of the tested population. Eight of these recruits had only slightly enhanced threshold of speech reception and could get entry at the time of induction as pointed out by Corliss (1953) that people with a speech loss of more than 15 dB but less than 30 dB in either ear have difficulty only with faint speech. Three subjects who had hearing loss of speech which varied from 38 dB to 50 dB could not have qualified for recruitment showing that the whisper and voice tests imparted were not sensitive enough to eliminate them. 102 had normal hearing and had less than 15 dB of hearing threshold at all frequencies and conformed to the standard of Glorig (1964) who suggested that pre-employment audiogram of recruits should not exceed 15 dB at 500, 1000 and 2000 cps.

The hearing defects in any population is usually either due to noise, the physical cause or any other organic causes like diseases of the ears. All the recuits hailed from villages and history noted depicted no aural assault before joining the service. During their average service, tenure of 3.2 months also, they were not put to much noisy environment and thus hearing loss in the present group of subjects was due to some ear disease suffered before induction. Actually 4 subjects had stated in the questionaire that they suffered from running ears in their childhood and the rest did not remember having suffered any ear ailments. Out of these 4 only one was aware of being hard of hearing and other 3 claimed themselves to be normal. This is in accordance with Coles (1964) who stated that high tone losses have to be quite severe before they have any effect on the identification of speech sounds and even then they only interfere with the reception of a few consonants such as 's', 'f' and 'th'. Unless hearing below 2000 cps is appreciably affected, as well as high frequencies, the deafness is not usually noticeable to the subject

All the recruits having defective hearing had higher hearing threshold at higher frequencies (3000 to 8000 cps). Black (1960) has shown that personal sounds like crackling of stones produced by walking over them or sound emanating while a person is urinating are predominantly of high frequency character and inferred that in order to hear them properly good hearing in the 2000 to 6000 cps zone is important. The detection of personal sounds is believed to be of importance to the fighting efficiency and safety of soldiers in such situations as sentry, ambush and fighting in conditions of limited range of vision (Jungle, thick undergrowths, wood etc). These recruits having high tone hearing losses were thus not suitable for the duties enumerated above.

This study also indicates that whenever the question of compensation for suffering hearing loss during service tenure will arise, all the recruits cannot be taken as having normal hearing at the time of induction. Also from safety and efficiency points of view all the recruits can not be employed for specialized duty where good hearing is obligatory. Thus it is necessary to collect complete preplacement audiogram to avoid future administrative difficulty and lack of efficiency. However, it does not appear to be feasible because of the prohibitive cost and time requiremnt. Hence it is suggested that screening audiometry at 500,2000 and 6000 cps may be introduced for recruits in the armed forces and this record may be maintained until the person retires.

The present study brings out that the whisper and voice tests are not adequate to eliminate recruits with high tone hearing loss and may allow a few inductees with hearing loss in speech frequencies as well.

SUMMARY

Audiometric study on 113 army recruits revealed that 11 recruits forming 10% of the tested population had hearing loss before induction. Three of these had 38 to 50 dB of hearing loss in speech frequencies as well. Even these three got entry into service showing that the whisper and/or voice test imparted were not efficient enough to eliminate such cases.

ACKNOWLEDGEMENT

The authors are thankful to Surg. Capt. MS Malhotra, Director Defence Institute of Physiology and Allied Sciences for his keen interest in the study, to Lt Col GL Dua for making arrangements and to the subjects for their fine cooperation.

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AUDIOMETRIC SURVEY OF ARTILLERY PERSONNEL

BY

Lt Col S.R.V. Kessar*

INTRODUCTION

Adverse effects of noise on hearing in man have been known for a long time. Goldsteine (1933) found the first reference to gunfire deafness in 1591. Fosbroke (1830-31) recorded his findings that blacksmiths as a group suffered from varying degrees of hearing loss. In 1864, someone who called himself "Volunteer" asked in Lancet "Can any one of your readers suggest a remedy for preventing, hearing loss by rifle shooting" (Glorig 1958). C.H. Parry as quoted by Burns (1968) described cases in which hearing was either temporarily or permanently impaired due to gun fire noise. In one incident Admiral Lord Rodney is described as being almost totally deaf for fourteen days following a broadside from his flagship "HMS Formidable" in the year 1872. In another instance an officer is stated to have been rendered permanently deaf by the discharge of nearby cannon in the battle of Copenhegan. Despite the historical allusions to the effects of gun-noise, the ill effects of such noise seem to have been till recently totally ignored or tacitly accepted. An attitute of bravado or "It can not happen to me" seems to prevail amongst the gunners. A degree of hardness of hearing tends to be accepted as the hall mark of the gunners whereas the gunners who are relatively resistant and seemingly unaffected are often quoted as examples justifying non-use of protective measures. (Coles & Rice 1965).

Following numerous reports of deafness due to gun-noise during and after the 1939-45 war, and with the general increase in awareness of the hazard, extensive investigations into the physical properties of noise and its effects on the hearing of those who are exposed to the hazard have been made. In this connection outstanding contributions made in the study of impulse noise by the Royal Naval Medical School, Alverstoke in collaboration with Institute of Sound and Vibration Research University of Southampton in U.K on one hand and the US Army Human Engineering Laboratory, Aberdeen Proving Grounds Maryland USA on the other, deserve special mention. It is only recently that the technical knowledge concerning the measurement of noise, relationship between physical aspects of the noise environment and the noise induced hearing loss has reached proportions that permit more or less definite conclusions between them.

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MATERIAL AND METHOD

The present Audiometric Survey was undertaken with a view to assess adverse effects of gun-noise on the hearing acuity of the artillery personnel in general and the gunnery crew in particular. The survey which was carried out in Aug-Sept 1972, included one Medium Regt, (130 mm guns) One Light Regt (120 mm Mortars) and one Fd Regt (120 pounder guns). All available personnel of the regiments have been included in the survey.

Three BEL (Bharat Electronics Ltd.) type KMK 222-A audiometers were used. The survey was carried out in field where neither audiometeric booths nor sound treated rooms were available. To overcome these handicaps a "Biological Calibration" of each audiometer under "Test Conditions" was carried out by performing audiometry on 10 "Normal Subjects" (20 Ears) selected from AMC personnel (Age group 17-24 yrs). Zero level for each frequency in respect of each audiometer was established by obtaining the mean value for 14 "Best Ears".

Initial audiometric screening with "Individual Pure Tone Sweep Check Technique" (Glorig 1965) employing 1 KHz, 4 KHz & 6 KHz frequencies 10 dB above the "Zero Level" was carried out. Subjects who heard all the tones were categorised as "NOT AFFECTED". Those who failed to hear any of the tones by either ear, were subjected to a thorough otological examination including a detailed evaluation of history of exposure to shell, bomb or mine blast, with a view to exclude any other detectable cause for hearing loss apart from the gun-noise. Subjects presenting with any evidence of aural pathology or where history of exposure to blast was available were excluded. In the remainder a pure tone threshold audiometry was carried out. Only those subjects who on audiometry revealed a hearing loss of 15 dB or more in either ear were categorised as "AFFECTED". Since none of the subjects included in the survey had been exposed to the gun-noise during the preceding 30 days or more, audiometric changes recorded represent Noise Induced Permanent Threshold Shift (NIPTS).

For purposes of analysis the personnel of each Regt have been divided into three main categories :-

(1) Officers :- This is a small hetrogenous group where on account of the nature of duties, noise exposure is variable and difficult to estimate.

(2) JCOs :- Again a comparatively small, hetergenous group, whose mean age (34.24 year) is significantly higher than other groups and noise exposure is uncertain and variable hence difficult to estimate.

(3) ORs :- Because of different trades, this category has been further subdivided into 3 groups according to degree of exposure to gun-noise.

"Group A" includes only gunnery crew who are exposed to the gun-noise from close quarters.

"Group B" includes such categories as Operator Radio Artsllery (ORA), Technical assistants (TA) MT drivers, etc who are ordinarily exposed to the gun-noise from a distance of 30 to 100 meters, but may occasionally, especially during actual combat, be exposed to the gun noise from close quarters, hence the noise exposure is variable and difficult to estimate in this group also.

"Group C" (Control Group) includes such categories as clerks, cooks, washermen, storemen, mess-waiters etc who are hardly ever exposed to the gun noise from a distance less than 400 meters and hence have been selected as controls. Since the gunnery crew (Group A) and the control group (Group C) belonged to identical social strata, ethenic groups, had identical nutritional status as well as almost identical exposure to small arms fire, the two groups ("A" and "C") are considered statistically comparable in all respects except one variable ie exposure to gun noise.

Age wise comparison of the gunnery crew (Group A) and the control group is given in Table 1.

Table I

Age wise comparison of Group "A" (Gunnery Crew) and Group "C" (Control group)

	Total No	Maximum Age	Minimum Age	Mean Age
Group "A"	262	32 yrs 4 months	17 yrs 1 month	23 yrs 2 months
Group "C"	239	36 yrs 2 months	17 yrs 4 months	25 yrs 7 months

Table II

Incidence of hearing loss in the Medium Regt

Category	Total Numbers	Normal Subjects		Affected Subjects	
		Number	Percentage	Number	Percentage
Officers	9	7	77.78	2	22.2
JCOs	9	4	44.44	5	55.56
OTHER RANKS					
Group A (Gunnery Crew)	86	48	55.81	38	44.19
Group	154	125	81.17	29	18 83
Group C (Control Group)	72	64	88.89	8	11.11

RESULTS

The term hearing loss as used here denotes an audiometric finding of which the subject may be unaware in contrast to "deafness" which implies subjective perception of disability.

In the Medium Regt (Table II) the incidence of hearing loss amongst gunnery crew (Group A) is 44.19% as compared to 11.1% in the control group (Group C). In Group B the incidence of hearing loss is 18.93%, approximately 50% higher than the control group.

In case of the The Light Regiment (Table III) the incidence of hearing loss amongst the gunnery crew is 57.14% as compared to 16.67% in the control group and 21.71% in Group "B".

In the Fd Regt (Table IV) incidence of hearing loss in Group A as well as Group B and the control group (Group C) is highest, being 69.57%, 26.32% and 23.81% respectively However, the data in respect of the Fd Regt is not considered reliable. Firstly, the regiment had come under heavy counter bombardment during Dec 1971 operations, as such history of exposure to Shell or Bomb blast was very common. Secondly, the audiometric survey was carried out at a high altitude area within a few days of the induction of the regiment to this area. The troops had not fully acclamatised with the result that their responses during audiometry were somewhat erratic and unreliable. In view of this the data pertaining to this regt has been excluded from further discussion.

The over all incidence of hearing loss in respect of the Medium Regt and the Light Regt (Table V) amongst gunnery crew (Group A) is 50.85% as compared to 14.1% in the control group.

Table III

Incidence of hearing loss in the Light Regt

Category	Total numbers	Normal Subjects Number	Normal Subjects Percentage	Affected Subjects Number	Affected Subjects Percentage
Officers	9	6	66.67	3	33.33
JCOs	8	2	25.00	6	75.00
Other Ranks :-					
Group A (Gunnery Crew)	91	9	42.86	52	57.14
Group B	175	137	78.20	38	21.71
Group C (Control Group)	84	70	83.33	14	16.67

Table IV

Incidence of hearing loss in The Fd Regt

Category	Total number	Normal Subjects		Affected Subjects	
		Number	Percentage	Number	Percentage
Officers	7	5	71.43	2	28.37
JCOs	6	2	33.33	4	66.67
Other Ranks :-					
Group A (Gunnery Crew)	69	21	30.43	48	69.57
Group B	133	98	73.68	35	26.32
Group C (Control Group)	63	48	76·19	15	23.81

Table V

Over all incidence of hearing loss in the Medium and the Light Regt combined together

Category	Total number	Normal Subjects		Affected Subjects	
		Number	Percentage	Number	Percentage
Group A	177	87	49.15	90	50.85
Group B	329	262	79 64	67	20.36
Group C	156	134	85.90	22	14.10
"A" Vs "C" P< 01					
"A" Vs "B" P< 01					
"B" Vs "C" P<.05					

This high incidence of hearing loss in gunnery crew closely approximates to other published figures. Flodgen (1945) reported an incidence of hearing loss of 58% amongst Swedish gunners, Murray & Reid (1946) found 50% of the Australian gunners affected, while Rai & Chaturvedi (1972) found hearing loss of varying degrees in 56.76% in gunnery crew of the Medium Regts surveyed by them.

With a view to assess the relationship of the amount of exposure to gun-noise with incidence of hearing loss, data regarding exposure to the number of rounds in respect of each gunnery crew was collected. However following a review of the data collected, and the available literature it is felt that a better guide to the amount of exposure to gun noise would be length of service for a number of reasons. Firstly no accurate record of the number of rounds fired by each individual was available, secondly the gunnery crew are exposed not only to the noise of their own guns which is usually incident at the ears at a grazing angle, but also to the noise of the adjoining guns of the battery which may be incident at normal angle (right angle), which is potentially more hazarduous. Lastly, with practice some of the gunnery crew learn to activate voluntarily the tympanic reflex, just before the firing of the gun which has a protective action (Coles & Rice 1965). This protective action is only available, when the firing is anticipated and as such is not applicable against noise enemating from the adjoining guns of the battery. Keeping this in view the subjects in group 'A' have been subdivided into three sub-groups according to length of service. (a) less than 5 years service. (b) between 5 to 10 years service (c) over 10 years of service. Out of 19 subjects with less than 5 years service, 4(21%) are affected. In 5 to 10 years service group out cf 121 subjects 54 (44.6%) are affected and out of 37 with more than 10 years service 32 (86.5%) are affected (Table VI). It is clear that with increasing exposure to gun-noise the incidence of hearing loss rises, reaching as high a figure as 86.5% in subjects with more than 10 years of service, a position which clearly calls for urgent remedial action.

Table VI

Incidence of Hearing Loss in relation to length of service (Group A)

Service group	Total number	Affected Subjects Number	Affected Subjects Percentage
Upto 5 years	19	4	21.00
5 to 10 years	121	54	44.6
More than 10 years	37	32	86.5

Table VII

Degree of Hearing Loss in relation to length of service in Group "A"
(Affected Subjects only)

Service Group	Total number	Hearing loss not exceeding 30 dB Number	Hearing loss not exceeding 30 dB Percentage	Hearing loss 30 to 60 dB Number	Hearing loss 30 to 60 dB Percentage	Hearing loss above 60 dB Number	Hearing loss above 60 dB Percentage
Upto 5 years	4	1	25	1	25	2	50
5 to 10 years	54	18	33.3	17	31.5	19	35.2
More than 10 years	32	11	34.4	9	28.1	12	37.5

It may be argued that these figures represent only an audiometric finding and do not necessarily indicate a hearing impairement amounting to disablement. However a further analysis of the audiograms of the affected subjects in Group 'A' shows that out of a total of 90 affected gunnery crew, in 30 (33.3%), the Hearing Loss is less than 30 dB, in 27 (30%). Hearing Loss is between 30 to 60 dB and in 33 (36.7%) the Hearing Loss is even higher than 60 dB in one or both the ears (Table VII) Analysing the audiograms in terms of criteria laid down for hearing impairement by CHABA (working group 48-1966). 21.1% show more than 15 dB average hearing loss at 500 & 2000 Hz. Further more the subjects belonged to a comparatively young age group (Mean age 23 years 2 months). As is well known the relationship between presbyacusis and noise induced hearing loss is arithimatically additive, it is certain that with the onset of presbyacusis in due course of time, more and more subjects with sub-clinical hearing loss will develop a more noticeable hearing disability.

Another factor that requires consideration, is that the present survey was carried out when the regiments were in field areas. As such most of the personnel who had reached the stage of clinical deafness had probably been down-graded to lower medical categories and posted out of the units. It is reasonable to assume that the real overall incidence of the deafness disability may still be higher. An idea of the magnitude of this problem in the armed forces, purely in economic terms, leaving aside the humanitarian considerations may be had from the fact that in United States, The Veterans Administration pays over 40,000,000 dollars every year for inservice incurred deafness disability (Parprrella 1967).

The mean audiograms in respect of the affected subjects of the three sub-groups of the Medium Regt according to length of service are given in Figure I. It will be observed that the maximum hearing loss is in the region of 6000 Hz and not 4000 Hz as is the case in industrial noise deafness. Gravendeel & Plomp (1959), Coles (1970) and Kryter (1970) have also emphasised that the maximum hearing loss is in the region of 6000 Hz following exposure to gun noise and not in the region of 4000 Hz.

FIG-1

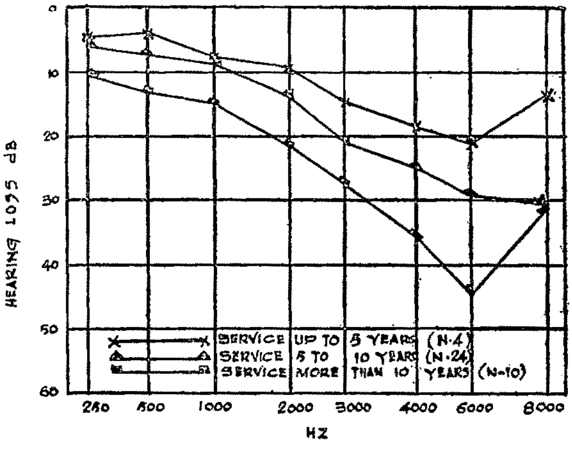

Fig I. Mean hearing loss of subjects of the Medium Regt according to length of service (Affected subjectes-Left Ear only).

The control group also shows a fairly high incidence (14.1%) of hearing loss even though this group was not exposed to gun noise from close quarters. This fairly high incidence of hearing loss could possibly be due to exposure to rifle noise during initial training and annual range classification over a period of years. It is well known that following exposure to rifle noise hearing loss is greater in the left ear as compared to the right ear, since the left ear is directly exposed to noise whereas the right ear lies in the "Sound Shadow" region of the head. The mean hearing loss for right and left ears separately in respect of the affected subjects in the control Gooup is given in Fig 2. It will be seen that the hearing loss in left ear is significantly higher especially in 4000 Hz, 6000 Hz and 8000 Hz. This further tends to support the assumption that the rather high incidence of hearing loss in the Control Group could possibly be due to exposure to rifle noise.

FIG. 2

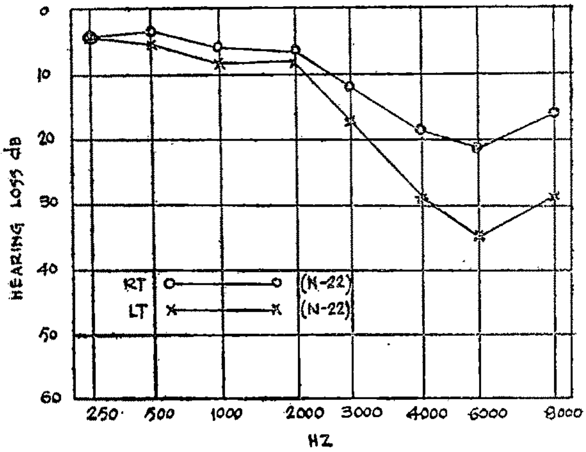

Fig 2. Mean Hearing Loss for right and left ears spearately in respect of Affected Subjects of the control group (Medium & Light Regt).

Evaluation of the noise hazard

It is relatively simple to study the various parameters such as sound pressure level (SPL), spectral content and exposure time of common industrial noises (continuous noise).

As a result of recent investigations an undoubted relationship between noise exposure level and hearing loss has now emerged. So much so that data obtained in the joint Medical Research Council and National Physical Laboratory investigation in UK (Burns and Robinson 1970) now enables one to specify with some precision the proportion of people working in a given occupational noise environment who will attain at a given frequency a particular hearing level after a specific number of years exposure to continuous noise. However on account of the brevity of the event (m/sec), measurement and evaluation of impulse noise has been the largest area of uncertainity, although in recent years the situation is improving considerably.

Gloring et al (1961) while discussing Damage Risk Criteria (DRC) recommended DRC for habitual exposure to continuous type of noise but regarding impulse noise stated that the limited knowledge regarding the impulse noise and its effects prevents any meaningful discussion of impulse noise and simply advised "No one should be habitually exposed to impulse noise of any considerable magnitude". CHABA (1965) laid down elaborate DRC for continuous noise but regarding impulse noise merely stated that "Impulse noise over 140 dB may be hazardous". On the other hand US Army TB Med. 251 (1965) reported that exposure below 140 dB may also be hazardous to some very sensitive ears.

Based on conclusions of independent British and American studies Coles et al (1968) described the technique of measurement of the impulse noise and also defined the various important parameters that must be considered in determining the potential hazard of the impulse noise. While commenting on the unsuitability of the conventional, visually read, needle type sound level meters for impulse noise measurement, Coles et al stressed that because of the brevity of the gun noise, it is essential that a suitable sound level meter is coupled to a Cathode ray ossilloscope and then the event is photographically recorded with a high speed camera. Using this technique the pressure/time history of the impulse is recorded and analysed in terms of peak pressure level, A duration, B duration and Rise time ete as given in Figure 3. The potentional hazard is then evaluated by reference to the graph shown in Figure 4.

FIG 3

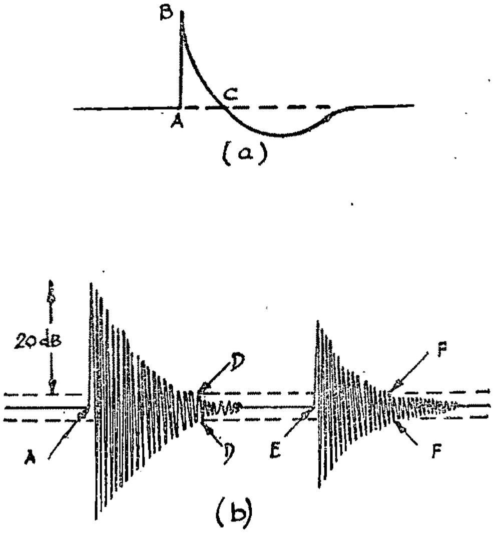

Fig. 3–Idealized evaluations of oscillographic waveforms of impulsive noises. Peak leve=pressure defference AB. Rise time difference AB: (a) A–duration=time difference AC. (b) B–duration= time difference AD (plus EF for example in the case of a relatively long–time reflection). Coles (1970).

FIG. 4.

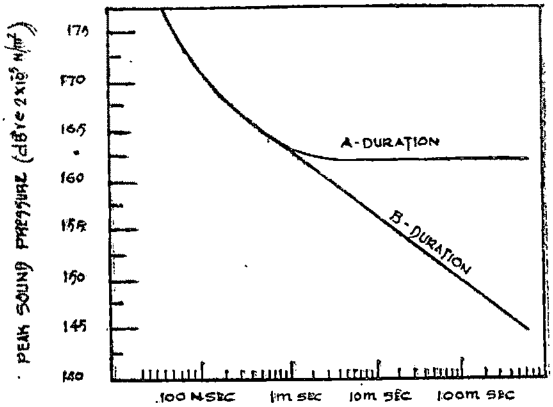

Fig. 4 Damage Risk Criterion for impulse noise (Coles 1970)

(a) Criteria are based on the recommendations of CHABA (1966) which accepted end of the day TTS-2, not more than 10 dB at 1000 Hz, 15 dB at 2000 Hz, & 20 dB at 3000 Hz in 50% of normal hearing persons exposed to the noise.

(b) In view of the greater variation in range of TTS to impulse noise the authors have applied the CHABA limits to 75th percentile ie below these limit 75% of exposed persons are not likely to suffer TTS-2 mentioned in (a) above.

(c) Criteria are based on repetition rates of 8 – 30/mt.

(d) Maximum number of 100 impulses per occasion and about 10 occasions per year.

Subsequently Ward (1968) has also proposed DRC for impulse noise, and his tolerable limits are very similar to those proposed by Coles et al. Ward further suggested 179 dB as the upper limit for any type of impulse noise.

Murray and Ried (1946) recorded 154 – 159 dB from .303 rifle, Coles and Rice (1965) reported 168 dB at firer's ear from British Self Loading Rifle and 184 dB from antitank gun. Kryter and Garnither (1965) have recorded 159 to 172 dB from various American weapons fired from the shoulder. Coles et al (1968) have reported the following data in respect of American M 14 rifle and British LIAI.

	M 14	LIAI
Peak pressure level at firer's ear	159 dB	161 dB
A duration (u sec)	270	300
B duration (m sec)	5	5

Rai and Chaturvedi (1972) employing an ordinary needle type sound level meter recorded 132 dB at 80 yards from 130 mm gun firing with full charge and arrived at the computed value of 164 dB at a distance of 2 yards. When compred to other published figures, 164 dB at two yards from 130 mm gun firing with full charge would appear to be too low. Although no reliable data for various types of artillery guns is available it would appear to be reasonable to assume that the gunnery crew are probably exposed to sound pressure levels of 180 dB to 190 dB, even beyond the upper limit of 179 dB recommened by Ward for any type of impulse noise or condition of listening. Need for instituting protective measures against the gun noise is obvious.

Protective Measures :–

Protective Measures may be discussed under two heads :—

(1) Individual Protective Measures
(2) Detection of susceptible subjects

Individual Protective Measures :–

Individual protective mesaures relate to the use of sound attenuating devices worn by those exposed to hazardouse noise levels. Ear muffs and ear guards (Ear plugs) have received considerable attention as a means of protecting the ear against noise. The attenuating characteristics of ear muffs and ear guards commonly used in industry are given in Figure 5. There devices interact in various ways with intelligibility of speech by the users, as the noise and speech signals are attenuated equally at any one frequency. Since signal to noise ratio would remain unaltered speech intelligibility would be expected to remain the same with or without the protective devices. However in practice in high level continuous noise, the speech intelligibility actually improves with ear plugs or ear muffs (Kryter 1970) because the speech and noise are reduced to a level at which ear is not over loaded thereby improving the speech discrimination. However, when the noise is of low level the intelligibility of speech is redueed by the devices because the speech is reduced to a level below the listeners threshold of hearing.

FIG. 5

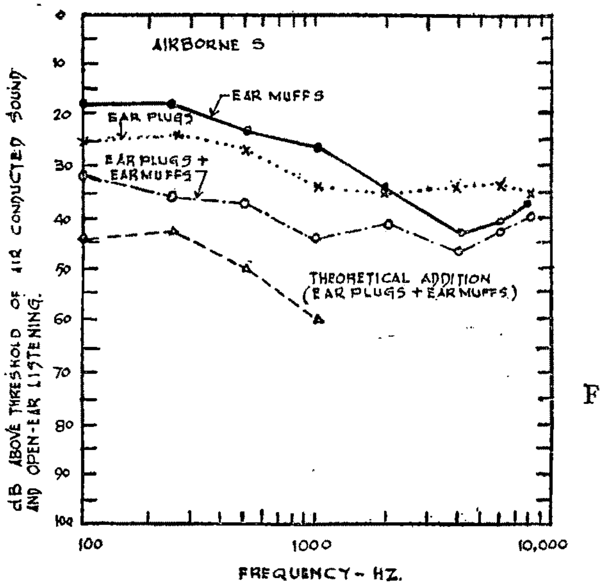

Fig. 5–Measured attenuation curves of (a) earplugs, (b) earmuffs, (c) earplugs and earmuffs together, (d) theoretical addition of attenuations in decibels provided by earplugs and by earmuffs (Kryter 1970).

Although the types of attenuating devices (Ear muffs and Ear guards) used against industrial noite are generally found to provide adequate protection against the gun noise, but unfortunately the problem of hearing low level sounds particularly speech becomes acute during the comparatively long noise free intervals between the impulses. This interference in hearing of speech sounds during the noise free intervals is not only annoying to the users but is also potentially hazardous for obvious reasons. Communication may be preserved by the use of a closed system using noise - cancelling microphones, and earphones mounted within the defender, together with a trailing wire or magnetic loop; but this solution is too expensive and too clumsy for many purposes. The ideal would be non-linear attenuating device which would let weak sounds past but would attenuate intense sounds. ERDE defender produced by Explosive Research and Development Establishment (UK) uses this principle and little speech intelligibility is lost. It incorporates a microphone and a peak limiting amplifier and ear phones built into an ear muff, so that sounds of about 95 SPL are heard normally but the amplifier attenuates the transmission of sound above this level. Although it is very efficient, it is rather expensive and cumbersome.

A much simpler means of achieving the same result, but with a slightly less sharp cut off is provided by the principle of non-linear increase in impedence offered by a small orifice (Ingrad & Ising 1967) where the originally laminar flow becomes turbulent at higher sound pressure levels. Based on this principle a modified V.51R ear plug (Sonex) has been designed and provisionally named Gundefender to emphasise its role (Forrest 1970). In this the core of the plug is removed and replaced by a disc of 0.13 mm steel skin pierced by a 0.64 mm orifice. The speech attenuation of the Gundefender is 5dB against 20 dB of unmodified V.51R (Figure 6). The increase in attenuation first becomes evident at about 110 dB and for impulses above 140 dB the increase is quite steady at the rate of 1 dB for 2 dB increase in the incident noise and at 190 dB the attenuation becomes 30 dB, same as for V.51R.

Gundefender has been tried in extensive field trials in the British Army and the results have been claimed to be very encouraging both in relation to speech communication and ear protection qualities. Cundefendar appears to be a highly suitable device for the gunhery crew and merits a trial.

FIG.6

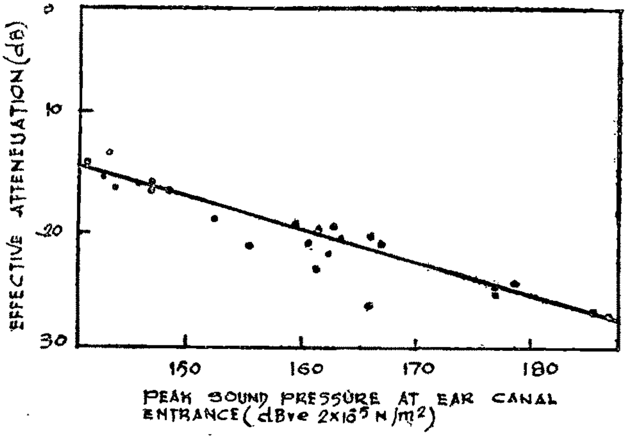

Fig. 6-Impulse attenuation from "Gundefender" ear plug as a function of peak sound pressure at the ear canal entrance. (Forrest-1970).

Detection of Susceptible Subjects :–

It has long been recognised that individuals vary in their ability to stay healthy. Bombarded by equal amounts of radiation some animals succumb several times as swiftly as others. Such variability between individuals is also found in hearing loss due to noise (Ward & Nelson 1969). Wide variation in susceptibility to impulse noise in paricular has been stressed by a number of workers. Kryter & Garnither (1966) while commenting upon the wide variation in susceptibility to impulse noise stated "We are particularly impressed by the great variability of threshold shifts exhibited by different ears as a result of exposure to gun noise. Indeed it appears that the distribution of sensitivity is bimodal, that is there are "Tender Ears". and "Tough Ears". Ward (1966) during a study of relationship of TTS to PTS emphasised the wide variation in susceptibility to impulse nocse. Coles & Rice (1965) have also stressed the wide variation in susceptility. Even in the case of Damage Risk Criteria (DRC) recommended by various committies and workers, the noise is deemed to be acceptable if the spccified hearing loss is limited to 50th percentile for ordinary industrial noise, but on acconnt of the marked variation in susceptibility to impulse noise, the impulse noise is deemed to be acceptable only if the specified hearing loss is limited to 25th pereentile.

In view of the obvious importance of predicting susceptibility, a test has long been sought by which it would be possible to predict susceptibility to noise. Since a number of factors are involved that influence the susceptibility it became clear to early investigators that the best chance to predict susceptibility to permanent hearing loss probably was embodied in study of Temporary Threshold Shift (TTS). The main advantage of TTS as a predictor of PTS is that it will depend upon much the same variables as those we assume for differences in PTS. There appears to be a general consensus that on average there is a close relationship between TTS & PTS as far as continuous noise is concerned. However Gravendeel & Plomp (1959) questioned this relationship between TTS & PTS in respect of gun noise, on the grounds that the average maximum permanent dip in the audiogram of 228 soldiers was at 5900 Hz whereas thirty six, 20 yrs old recruits showed an average maximum temporary dip at 4600 Hz. Kryter (1970) while commenting on this has pointed out that the small difference in the locus of the dip for two groups could well be attributable to the small size of the recruits, some natural selection factor in the older soldiers, and difference in the types of the gun noise to which the two groups have been exposed, and concluded that other things being equal the ear most susceptible to TTS, is most likely to suffer permanent damage. Coles et al (1968) are also of the opinion that on average a TTS if repeated often enough may become PTS of similar magnitude. Ward & Nelson (1968) have also concluded that the best estimate of the susceptibility to permanent damage will be provided by a TTS test using the specific noise.

Beginning with Peyser (1940) a number of workers have proposed susceptibility test involving study of TTS to predict PTS. Most of these tests pertained to the common industrial noises. In 1967 Elwood described a test which has been used in the British Army to dctect susceptible individuals. In this test TTS–2 (temporary threshold shift at 2 minutes) of 20 dB or more at 3 KHz, 4 KHz, 6 KHz and 8 KHz is used as the criterion for susceptibility. In one study out of 151 men exposed to rifle fire, (160 dB) fired at 5 seconds intervals in groups of 20, 40, 60 rounds, using this criterion Elwood found 37 susceptible to 20 impulses, 29 susceptible to 60 impulses, 13 susceptible to 120 impulses and 72 did not reveal any significant TTS even after exposure to 120 impulses. It is clear that there is a marked individual variation in susceptibility to impulse noise provided the assumption that TTS is a reliable guide of PTS, is coorect. However, no published reports regarding a longitudinal study co-relating the TTS to PTS in the same groups of individuals is available. It is felt that a study of this nature for which an opportunity still exists in our Armed Forces would be highly usefull.

SUMMARY

Results of audiometric survey of three Artillery Regiments are presented. The survey revealed that 50.85% of the gunnery crew had hearing loss of varying degrees as compared to an incidence of 14.1% of hearing loss in the controls. The incidence of hearing loss in the gunnery crew rises with length in service, reaching as high a figure as 86.5% in the gunnery crew with more than 10 years of service. Methods of evaluation of the gun-noise hazard and preventive measures are briefly discussed.

ACKNOWLEDGEMENTS

It gives me great pleasure to acknowledge, the help so willingly given by Lt Col B Singh, Otologist MH Jullundur Cantt and Major K Gopalan, Otolgist MH Pathankot in carrying out the audionetric survey.

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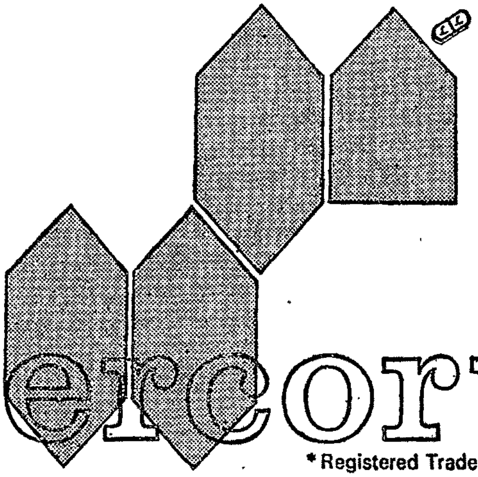

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DEVELOPMENT OF PARANASAL AIR SINUSES

(A roentgenographic planimetric study)

BY

Lt Col A.K. Ghosh*

INTRODUCTION

Turner (1901) was the first worker to describe the development of paranasal sinuses of English skulls. Since then Schaeffer (1936), Murai (1937), Naatanen and Paatero (1938) and Stern (1939) studied the development of paranasal sinuses of various nationalities. Koranne et al (1963) established the average measurements of only maxillary sinus in a study of 25 Indian skulls. Except Turner (1901) and Schaeffer (1936) no author has described the complete development of paranasal sinuses and even these studies are not correlated.

MATERIAL AND METHODS

One hundred randomly selected individuals of eastern India in the age groups 3 to 4, 7 to 8, 11 to 12, 15 to 16, 24 to 25 years of both sexes in equal numbers were included in this study of development of paranasal air sinuses. It was made sure that the subjects had a normal otolaryngological finding and there was no history of paranasal sinus infection in the past. The dental formula of each was recorded and the type of dentition was also noted. Roentgenograms of the skull were taken in occipitomental and lateral positions. The sinus areas were measured by a planimeter. The planimeter is an instrument to measure the areas of irregular figures. The paranasal sinuses were studied for their size variation and rate of growth. An attempt was also made to correlate their development with that of skull as a whole, size of the orbit and eruption of teeth. In this study frontal, maxillary and sphenoidal sinuses were considered. Ethmoidal sinuses, however, were excluded from this study due to obvious radiological technical difficulties.

OBSERVATIONS AND DISCUSSION

Detailed findings have been tabulated in tables 1 to 4.

1 The size variations of frontal, maxillary and sphenoidal sinuses increase with age (Figs 1, 2, 3, and table 1).

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2 Maximum asymmetry between right and left frontal sinusess is at 11 to 12 years in both sexes and maximum asymmetry of maxillary sinuses is at 3 to 4 years and decreases with age (Table 1).

Table 1

Mean areas of sinuses at different age groups (all figures in sq cm)

Age group	Frontal sinus			Maxillary sinus			Sphenoid sinus
	Rt	Lt	Lat	Rt	Lt	Lat	Lateral
MALES							
3-4 years	0.50	0.91	0.41	0.69	1.39	2.62	0.80
7-8 years	0.54	0.48	0.46	2.12	2.32	4.37	1.77
11-12 years	1.65	3.61	1.26	3.87	4.02	10.11	3.63
15-16 years	5.34	4.67	1.50	6.03	5.04	8.44	5.48
24-25 years	7.14	7.62	2.09	5.73	5.73	9.62	7.48
FEMALES							
3-4 years	0.37	0.63	—	0.52	1.73	1.41	0.34
7-8 years	0.92	0.55	0.42	1.69	1.87	5 29	1.38
11-12 years	1.11	1.65	0.68	4.76	4.09	7.71	3.56
15-16 years	4.27	4.35	2.16	4.34	4.50	9.00	2.74
24-25 years	4.05	4.03	1.55	4.56	4.22	9.53	6.13

3 Maximum rate of growth of frontal sinuses is at 15 to 16 years and continues upto 24 to 25 years in both sexes (Fig 1 and table 2).

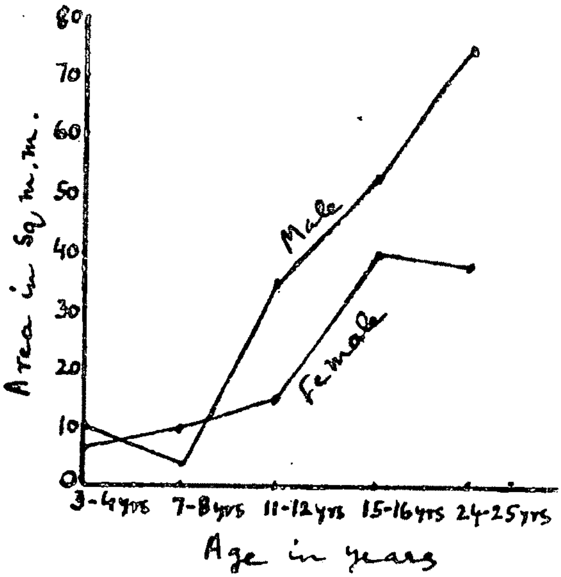

Fig. 1 Graph showing the rate of growth of frontal sinus.

Table 2

Relation between Frontal, Maxillary and Sphenoid sinuses (All areas in sq cm)

	LATERAL VIEW			RATIO				OCCIPITOMENTAL VIEW	
Age group	Frontal sinus	Maxillary sinus	Sphenoid sinus	Fr.	Max	Sph.		Frontal siuus Rt + Lt	Maxillary sinus Rt+Lt
Males									
3–4 years	0.41	2.62	0.80	1	6.5	2		1.5	3.0
7–8 years	0.46	4.37	1.77	1	11.0	4		1.0	5.4
11–12 years	1.26	10.1	3.63	1	9.0	3		5.2	7.8
15–16 years	1.59	8.44	5·48	1	5.0	3.6		10.0	10.0
24–25 years	2.09	9.62	7.48	1	4.8	3.7		14.7	11.4
							Mean	6.5	7.5
							SD	5.2	3.0
								r=0.99	
FEMALES									
3–4 years	–	1.41	0.34					1.0	1.2
7–8 years	0.42	5.29	1.38	1	13	3		1.4	3.5
11–12 years	0.68	7.71	3.56	1	12	6		2.7	8.8
15–16 years	1.16	9.03	2.74	1	4.3	1.3		8.6	9.0
24–25 years	1.55	9.52	6.13	1	6.7	4.3		8.0	8.7
							Mean	4.3	6.2
							SD	3.2	3.2
								r=0.78	

4 The rate of growth of maxillary sinuses is at their peak at 11 to 12 years and continues to grow up till 24 to 25 years in both sexes (Fig 2 and table 2).

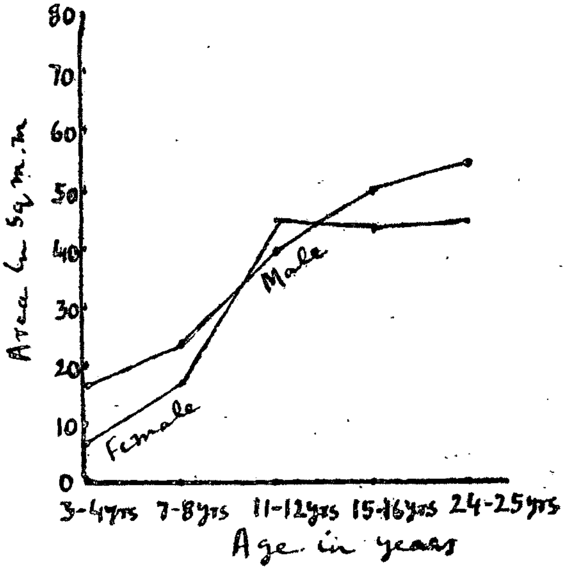

Fig. 2 Graphs showing the rate of growth of maxillary sinus.

5 The rate of growth of sphenoidal sinus continues till 24 to 25 years in both sexes (Fig 3 and table 2).

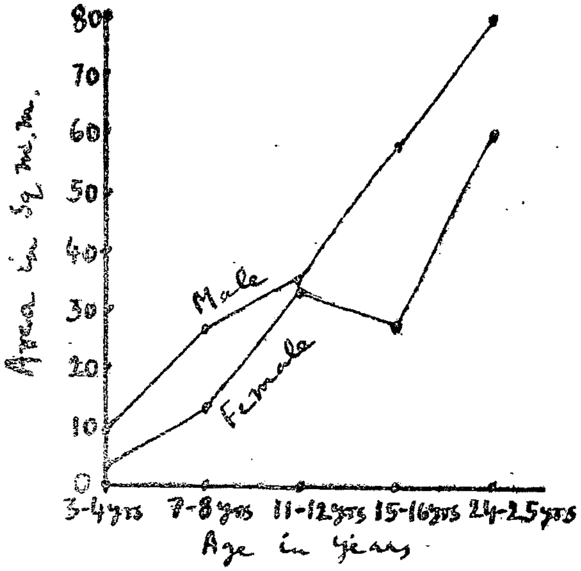

Fig. 3 Graph showing the rate of growth of sphenoid sinus.

6 There is significant correlation between the development paranasal sinuses with that of skull (Table 3).

Table 3

Relations of the paranasal sinuses with area of the skull in occipitomental position (All areas in sq cm)

Age group	Frontal sinus Rt+Lt	Maxillary sinus Rt+Lt	Area of skull in AP view	
MALES				
3-4 years	1.5	3.08	161.7	The coefficient of correlation between maxillary sinusess and the area of the skull is 0.989, and frontal sinuses is 0.80.
7-8 years	1.0	5.44	192.3	
11-12 years	5.2	7.89	207.1	
15-16 years	10.0	10.0	225.4	
24-25 years	14.7	11.4	233.2	
Mean	6.5	7.5	203.6	
SD	5.2	3.0	25.62	
FEMALES				
3-4 yeass	1.0	1.25	179.6	The coefficient of correlation between the area of the skull and maxillary sinuses is 0.595 and frontal sinuses 0.943.
7-8 years	1.4	3.56	174.9	
11-12 years	2.7	8.85	175.9	
15-16 years	8.6	9.00	215.4	
24-25 years	8.0	8.78	216.0	
Mean	4.3	6.24	191.8	
SD	3.29	3.26	19.98	

7 The orbits are twice bigger than the maxillary sinus after 11 to 12 years in both sexes (Table 4).

Table 4

Relations of paranasal sinuses with dentition and area of the orbits.
(All area in sq cm)

Age group	Frontal Sinus Rt+Lt	Maxillary Sinus Rt+Lt	Area of the orbits Rt + Lt	Total No of teeth	
MALES					
3-4 years	1.5	3.0	14.9	20	The coefficient of correlation between the areas of the orbits and the maxillary sinuses is 0.906 and frontal sinuses is 0.721.
7-8 years	1.0	5.4	19.2	23	
11-12 years	5.2	7.8	19.0	28	
15-16 years	10.0	10.0	20.0	28	
24-25 years	14.7	11.4	21.4	30	
Mean	6.5	7.5	18.9		
SD	5.2	3.0	2.17		
FEMALES					
3-4 years	1.0	1.2	15.6	20	The coefficient of correlation between the areas of the orbits and the maxillary sinuses is 0.824 and frontal sinuses is 0.860.
7-8 years	1.4	3.5	17.4	23	
11-12 years	2.7	8.8	18.4	28	
15-16 years	8.6	9.0	20.2	28	
24-25 years	8.0	8.7	21.6	31	
Mean	4.3	6.24	18.6		
SD	3.2	3.24	2.27		

ABBREVIATION

SD – Standard Deviation

8 There is definite realtionship between the development of paranasal sinuses with the eruption of molar teeth and the rate of growth is faster when the molars are erupting (Table 4).

SUMMARY

A study of the development of paranasal air sinuses by rocntgenographic planimetric method of East Indian subjects has been made and the findings described.

ACKNOWLEDGEMENT

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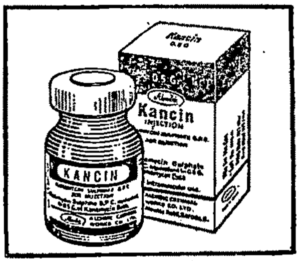

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PARANOID DISORDERS
A CRITICAL EVALUATION WITH SOME COMMENTS ON THOUGHT PROCESSES AND NEUROPHYSIOLOGICAL CORRELATES

BY

A.V.N. Rao*

INTRODUCTION

The subject of paranoid disorders is of great interest and importance in the field of clinical psychiatry and yet one finds very little added to the furtherance of our knowledge in these conditions except what is already written in the well-known modern text-books and an occasional article in medical journals. Apart from the acute paranoid psychosis developing in persons consuming amphetamine or its congeners (3), there is once again little that helps us to understand the possible pathways in which paranoid ideation originates and manifests in the human organism. The ubiquity of paranoid disorders, of varying intensity and duration with equally varying social, clinical and prognostic significance, is recognised, occuring as they do among those past 30's, in alcoholics, in those belonging to the minority groups, in some who are partially deprived of one of their special senses, hearing in particular, in those who are victims of disfiguring deformities and in patients suffering from other psychiatric disorders, funcitonal as well as organic (18).

Occasionally paranoid ideation spreads beyond the person affected. Among psychiatric disorders this resembles a communicable illness comparable to the infectious diseases of internal medicine. We read reports, few however, of *folie a deux, a trois, a quatre* and ad infinitum when the situation becomes a raging public hostility of colossal proportions, involving groups of people holding different political or religious opinions or belonging to the less or highly pigmented sections of humanity (8). From time to time whole communities seem to respond to real or imagined threat with something approaching paranoid panic. The word paranoid has such a wide connotation that during war each belligerent country calls its enemy paranoid.

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Paranoia and paranoid states together make up according to Tyhurst about 10 per cent of hospital admissions. These admissions represent more the age at which paranoid reactions become socially intolerable than the age at which they begin to be recognisable. Female incidence is more than twice that of male. More patients come from urban than from rural areas. Reliable statistics are hard to come by because paranoid reactions are often regarded justifiable and reasonable even in normal persons (29).

The questions that readily crop up in our minds are : are we dealing with a social problem based on ethology, culture and child-rearing practices, a neuropsychiatric problem with a distinct organic pathology irrespective of the types of noxae affecting certain still undefined parts of the human brain or a purely psychogenic disorder based on trauma attributable to communication failure between human individuals, either inadvertently as in day to day living or purposefully during brain-washing and indoctrination ? If we are dealing with a breakdown of well-established conditioned reflexes in the genesis of paranoid ideation and behaviour, surprisingly little is mentioned in this regard in Pavlov's work on the dogs that suffered in the Leningrad flood (20). Paranoid ideation is probably a human privilege.

A more significant and pressing problem in need of careful study is paranoid ideation which seems to spring so often in all human organisations based on an established hierarchy for purposes of administration. It would be interesting to know the relative ease and frequency with which paranoid ideation originates under various forms of government.

There is some evidence that paranoid reactions and developments are not infrequently found among the blood relatives of schizophrenics (18). More knowledge is accumulating about the behaviour of these patients outside the mental hospital milieu. Hence, information gained from recent studies in schizophrenia are relevant to the understanding of paranoid disorders as well. The offspring of schizophrenics seemed more creative, original and religious, while pedigree studies point to an association between schizophrenia, musicality and social or political success (10, 14, 16). The thinking processes of parents of schizophrenics have characteristics distinct from those of others (30). The term "lateral thinking" applied to this is considered the basic essential of all creative work, lifting people out of the normal rut of Aristotelian logic (4, 16). This may, in a sense, be called paralogic. Freud referred to this when he wrote of primary process in respect of dreams whereas our waking thought process is secondary. Primary process makes use of symbolism, mysticism, superstition and magic, and is the basis in day to day existence of humour, wit, satire and paradox (1). It is just unfortunate that in the schizophrenic primary process thinking appears to go haywire and what happens to the healthy man in his dreams happens to him when wide awake. Paranoid is a word of Greek etymology meaning literally "reasoning beside itself". Much more needs to be learnt about the thinking processes in the pre-schizophrenic and the paranoid and the actual relation between lateral thinking and primary process of Freud. Both types of thinking are needed, to be sure, in view of their flexibility and novelty conducive for creative thought.

It is now realised that sickness has wide implications. Newer concepts speak of family pathology and the sick society (5, 12, 17, 26). The patient can be viewed as the scape-goat of the family or even of the society. Asking one to adjust to such a society is tantamount to driving him sick. Adjusting, then, will not be a sign of mental health but of ill-health. One may not agree *in toto* with what Szaz writes on mental illness being a myth but it certainly stimulates one to revise one's views and doubt if we are dealing with diseases in the medical model or something else (28). It is not sufficiently realised that ambiguity or equivocation leading to a failure or distortion of commnication between individuals is an important factor in the genesis of paranoid reactions. Equally important is the personality of the affected individual. Who, of us, has not, at some time in our lives, felt paranoid ?

HISTORICAL ASPECTS

The history of delusional insanity is a long one. Heinroth in 1818 thought it was a disorder of the intellect. In 1845 Griesinger described it as an intellectual disorder secondary to affective disturbance. We owe the term paranoia to Kahlbaum, in 1863. Krafft-Ebing went further stating that the illness was characterised by systematic delusions occuring in shy, solitary persons, with neither complete recovery nor complete deterioration. Magnan, calling the illness *'delire chronique a evolution systematique'* described four stages ; (1) the hypochondriacal stage with subjective analysis, (2) persecution stage, where suspicions turn to beliefs, (3) transformation stage, with delusions of grandeur and (4) rarely mental deterioration. Hallucinations may occur in the persecutory stage. Memory and general intellectual qualities remain intact. As early as 1877 Laseque and Falret described *'folie a deux'* wherein two persons live in comparative seclusion and develop the illness. In 1908 Gierlich and Friedman noted abortive forms of the illness: *'formes fruste'*, which were precipitated by situational stress, showing periodic occurance. Bleuler, in 1912, brought some order, emphatically stating that the illness is the result of faulty perception and interpretation, suspicions are absent in the grandiose forms and any alteration of affect is secondary. He recognised a constitutional predisposition in all these patients. Freud described in 1896 paranoia as being the product of defence mechanisms on par with hysteria. In 1911 he developed the psychopathology of these states as based on homosexual fixation and failure of repression. Kraepelin, 1919-21, distinguished paranoia, paraphrenia and paranoid schizophrenia: paranoia, an illness with chronic, fixed delusions and no hallucinations; paraphrenia, an intermediate illness without the deterioration of schizophrenia. Kolle in 1931 stated that the illness resembled schizophrenia symptomatically and genetically. In 1943 Cameron brought out the concept of pseudo-community and its probable relationship to a failure in early life to develop basic trust. Adolf Meyer in 1951 stated that all paranoid states are transformations of personality, and while reason is preserved it is side-tracked. He too stressed the appearance of the illness in sensitive types, characterised by systematised delusions and self reference with at times retrospective falsifications. He also noted megalomanic developments or deterioration, with occasional dangerous and antisocial reactions. In 1961 Kay and Roth described late paraphrenia, in patients over 60, females preponderating, more among unmaried peole, with conspicuous auditory hallucinations but preservation of memory, personality and intellect and of a chronic, unrelenting course (15). A rather important finding was that of Post; also in 1961, who described persistent persecutory states of the elderly (19). He did not find much familial incidence of schizophrenia, on the other hand there was more familial incidence of affective disorders. Only a fraction of these cases showed any resemblance to schizophrenia. There was no remarkable change in affect or personality. He agrees that these patients, on the whole, in spite of their body build suggestive of an affective disorder, presented partial or incomplete manifestations of schizophrenia, responding favourably to phenothiazines.

Cervantes (1547–1616) is famous the world over as the author of a literary masterpiece, Don Quixote. Herein one finds full length descriptions of Kraepelinian parancia (6).

PSYCHOPATHOLOGY

The pre-morbid personality of a paranoid patient is said to present characteristic features : sensitive, suspicions, jealous, ambitious, shy, prudish, dreamy, impractical, selfish, vain and significantly at times none of these. They may also be rigid and uncompromising, humourless, aggressive, resentful of discipline and sensitive to slights. They cannot safely be made fun of. The role of inferiority feeling is important in the genesis of the illness. Homosexuality is of variable significance, however. Social difficulties, isolation and ostracism in a proud and suspicious person act in vicious circles. This picture to a great extent rings true of a schizoid personality, but many overt schizophrenics need not necessarily have had pre-morbid schizoid personalities.

Projection, one of the mental mechanisms and of biological protective value to the human organism, helps in ego defence. It is of supreme importance in paranoid disorders. It may be regarded as abnormal when a sense of proportion is lost, it is persistent, or becomes habitual, gaining influence on the life of the individual and his behaviour. In such cases a particular idea is over-valued or the motives of others or actual events misinterpreted in spite of evidence to the contrary. Such phenomena are called ideas of reference. Side by side, the important ego function of reality testing becomes impaired, facilitating formation of false beliefs. Caution develops into suspiciousness, fear and insecurity are expressed in such disguises as pettiness, never-ending complaints, cynicism and selfishness. Most of these people blame the environment for their dissatisfactions with self. They are unable to admit their faults to themselves, A rebellious and superior attitude may for sometime help to bolster self-esteem. However, that prevents a realistic but intolerable self-evaluation.

The delusions held are commonly referred to events that are not inherently impossible, such as being poisoned or beaing a bastard. Instances of projection are common in actual or would-be sexual miscreants whether auto-erotic, homosexual or illicitly heterosexual, who believe themselves to be spied upon or that people can tell by looking at them that they are guilty. The social milieu, as well as the mores existing at any particular time and place, obviously influence such reactions. Freud pointed out that paranoia arose when homosexual trends escaped repression. This theory is not universally accepted. A large number of overtly homosexual persons do not develop paranoid delusions and an equally large number are paranoid without being homosexual.

A deficiency of basic trust would appear to be necessary for the development of paranoid trends.

Depression, to the extent of possible suicide, is not uncommon when the paranoid patient is overwhelmed by an environment with which he is unable to cope. His failure, in the litigious forms of the illness, in attempts to seek redress results in further feelings of injustice. However, in any particular case, all may not be delusion : each case needs careful investigation from various angles.

A fairly large number of paranoids do not marry, while many are divorced. Reasons for this could be that they are insufferable to their partners, being hostile and jealous, or on Freudian hypothesis their homosexual trends - made them unsuited for marital harmony.

PATHOPHYSIOLOGY

The appearance of paranoid colouring in most functional psychoses as the age of the patient advances is a well-recognised phenomenon. The most frequent form of schizophrenia in the middle-aged and the elderly is of the paranoid type. Delusions of paranoid nature are determined by the predominant affect in the affective psychoses Chronic deafness or any physical defect is associated with paranoid developments in some cases, but these are evidently determined by the feelings of inferiority engendered in the handicapped victims.

An acute impairment of cognitive functions, as occurs in subacute delirious states and symptomatic psychoses of varied aetiology, seem to favour development of paranoid ideation, but without systematisation or persistence. Hallucinations, when present, tend to be poorly formed. An altered level of consciousness is an important clinical concomitant in these cases, and the neuropathology lies in the ascending reticular activating system and its non-specific thalamocortical projections.

Paranoid colouring of a variety of organic dementias is a known clinical phenomenon. Paranoid psychoses occuring in the chronic epileptics, usually of temporal lobe pathology, are clinically indistinguishable from paranoid schizophrenia (25). An interesting feature is that this syndrome can arise in an individual who has no premorbid paranoid or schizophrenic trends in his personality, or family history of such illnesses. Skottowe prefers to call these paranoid psychoses dysrhythmic paraphrenia and he concludes, "it appears that a severe paranoid psychosis can be mediated essentially by the occurance of specifically located cerebral lesions, though the content of the delusions and hallucinations will reflect, of course, the patient's experience of life and his present social situation" (24). Amphetamine addiction and chronic alcoholism are other examples where paranoid psychoses develop Rare disorders such as vitamin B_{12} deficiency (11, 27) or encephalopathies of remote origin including those associated with bronchogenic carcinoma (7) may occasicnally be heralded by a paranoid disorder.

An important conclusion can be drawn from these correlations of clinical findings and brain pathology where there is no alteration in the level of consciousness in contrast to the acute brain syndromes. Certain brain mechanisms seem to be partcularly vulnerable, with advancing age, to the effects of specific biochemical or bacterial toxins. Excitation or inhibition of such areas releases paranoid ideation that is clinically indistinguishable from its functional counterpart. If bilateral temporal lobectomy in some cases is followed by failure to experience the characteristic hallucinations with mescaline or LSD one is led to conclude that the presence of intact temporal lobes and their connections are essential for hallucinatory manifestations in man (22, 23). If the capacity of an individual for normal dreaming is lost after bilateral frontal lobotomy an inevitable conclusion is that intact frontal lobes with their normal connections are a prerequisite for dreaming (9). Further, it is signifi- cant that late paraphrenics respond well to frontal leucotomy (21). Only further research and understanding of the inter-relations of the frontal and temporal lobes of the human brain can elucidate the possible neuropathological correlate of paranoid ideation.

PROBLEMS OF DIAGNOSIS

The International Classification of Diseases, Eighth Revision, Section V comprises 'Mental Disorders', and 'includes all forms of mental disorder, even though associated with or secondary to physical conditions' (13). Paranoid schizophrenia is to be classified under schizophrenia ; the group Paranoid States includes paraphrenia and paranoid reactions of old age ; acute paranoid reactions, however, are to be classified in the group of Other Psychoses.

Paranoid traits are observable in paranoid personalities who are from time to time liable to paranoid reactions in adverse circumstances : these are to be regarded as biological functions of self-defence. Such are the social cranks, faddists and eccentrics. These short episodes of paranoid rcactions are comparable to reactive depressions or neuroticism, and such attributes of personality are normally distributed throughout the population. It is thus seen that like depression which is a symptom as well as a clearly defined disease entity as in depressive psychosis or endogenous depression, paranoid disorders can be symptoms based on personality attributes of the individual or endogenous as in paranoia or paranoid schizophrenia.

In the diagnosis and differential diagnosis of paranoid disorders the following conditions, as far as possible, should be distinguished :

(a) Paranoid personality

Typically an irritable person, hypersensitive by nature, adopting more or less delusional approach to other persons and to life in general, all present throughout adult life and often commencing with the onset of adolescence. This may aptly be called a chronic paranoid state. Paranoid reactions are easily precipitatated if there is much predisposition, while largeerstresses would be required if the predisposition is weak.

(b) Paranoid reaction or Paranoid state

These are characterised by the persence of delusions, but lack the logical systematisation that is such an essential part of paranoia. At the same time there is no progress of the condition towards bizarre fragmentation and deterioration that are pathognomonic of schizophrenia. They may be short-lived or chronic. They may be associated with much anxiety. Depression to the extent of suicide is not unknown. Four clinical types are described : (1) jealous, (2) erotic, (3) grandiose and (4) persecutory, the last being the common est.

These reactions are developments that are understandable from a full knowledge of the situation and the personality of the patient. Incomprehensibility at any stage would point to a psychosis, probably schizophrenic in nature.

(c) Symptomatic paranoid states

These are secondary or incidental to a disturbance of cerebral metabolism. The conditions may be acute delirious states or chronic brain syndromes, amphetamine intoxication or effects of chronic alcoholism. Any incompetent brain, preventing a realistic grasp of what is going on in the environment or within oneself, is conducive to this condition. These reactions may be self-limited or prolonged depending on the nature of the damage to the brain. Many cases of dementia, including G.P.I. are associated with paranoid delusions, but they are seldom well sustained.

(d) Paranoid ideation as part of other functional psychoses

These are not uncommon : in depression and mania, and in schizophrenia.

(e) Chronic paranoid psychosis

Schizophrenia-like psychoses of chronic temporal lobe epileptics may be indistinguishable from paranoid schizophrenia, but for the history of epilepsy. These psychoses are termed dysrhythymic paraphrenia by Skottowe. It should be noted also that chronic deteriorated schizophrenics are liable to suffer from epileptic seizures. Occuring for the first time at a late age, they may closely resemble paranoid schizophrenia. Sometimes the psychosis is episodic in its course.

(f) Paranoia

As described by Kraepelin, paranoia is a very rare disorder. The essential diagnostic features of this condition are : great systematisation of delusions, with no disturbance of the patient's relations with reality. They are rarely brought to the notice of psychiatrists or admitted to mental hospitals. The condition may remain as 'encapsulated paranoia' and may in no way interfere with the patient's everyday life. Many people may not notice anything abnormal about these patients. There are no hallucinations at any stage of the illness and they do not deteriorate as the schizophrenics do.

(g) Paranoid schizophrenia

Of all the forms of schizophrenia, the paranoid type is the least variable and often persists true to type. Primary delusion is the crux of the diagnosis, followed by secondary elaborations and misinterpretations lasting for years. Deterioration is, however, late.

Many of these patients lack previous paranoid personality traits or even schizoid personality. However, if proper history is available, some paranoid schizophrenics might have had a catatonic form of the illness when young.

Wernicke, in 1906, described the 'delusions of explanation', which are props in the perplexity and distress suffered by the patient, providing him some stability in a world shaken up both internally and externally. The effects of primary delusions are supported by illusions and hallucinations, usually present from the beginning of the illness. Delusions concern magical forces, are usually persecutory and the patient's explanations are irrational. With increasing personality deterioration, the logicality of delusions becomes poorer and verbal expressions take on neologistic flavour.

Auditory hallucinations are of a threatening or accusatory type.

In the acute forms of the illness strong affective elements of fear, bliss or rage are not uncommon, but in general the paranoid schizophrenic lacks the intense drive for achievement which is part of paranoia. They are less sociable, less industrious and less restrained by social law and custom than the patients belonging to other paranoid categories. Acting-out is not uncommon and they may at times be homicidal. An important point of differential diagnostic significance is that in schizophrenia the patient gives up the struggle for self-esteem whereas an active struggle is sustained for years in paranoia. As time passes, any affect the patient possessed earlier on becomes flattened or "burnt out".

When paranoid ideas in a patient become bizarre and the mood capricious or incongruous, disorders of volition or psychomotor peculiarities in the form of grimacing, odd mennerisms and looking up in a bewildered way appear, one is dealing with paranoid schizophrenia.

Paraphrenia and late paraphrenia, for all practical purposes, can be included under paranoid schizophrenia, considering the difficulties involved in differentiating them from schizophrenia.

It is absolutely necessary to make sure of the presence of delusions, in a suspected paranoid disorder, and exclude the possibility of malicious relatives trying to "put away" the patient in order to acquire his property. Relatives have to be interviewed to enable a comprehensive understanding of the problems at issue.

History of alcoholism or addiction to drugs of the amphetamine group is important in diagnosing symptomatic paranoid psychoses, while conditions associated with disturbed brain metaeolism need systematic investigations along neurological lines. Their paranoid colouring, then, would be of secondary importance.

Family history is helpful. Paranoid personalities and paranoid reactions are not uncommon among blood relatives of schizophrenics. However, the incidence of schizophrenia among relatives of paranoid patients is only half that recorded in the families of schizophrenics.

If a chronological study of the development of the illness points to the presence of social and psychogenic precipitants acting on a touchy or sensitive pre-morbid personality and the clinical examination excludes symptoms suggesting schizophrenia, one is in all probability dealing with a paranoid state or reaction. Failure of response to phenothiazines and E.C.T. less in such cases would be a further point in favour of diagnosing paranoid state.

Diagnostic problems in any case presenting with paranoid symptoms are at times insuperable despite the clear-cut pictures that are drawn in the text-books. In a particular patient it may be impossible to prove the appearance of primary delusions or even the presence of auditory hallucinations. Only a painstaking cross-sectional study together with a longitudinal one can be of help.

MANAGEMENT

A thorough physical examination of the patient at the outset is imperative in order to exclude possible organic basis for the initiation or perpetuation of the paranoid state Undue indulgence in alcohol may be an important factor; so also the illicit consumption of amphetamine-like substances, apparently at one time prescribed for weight-reduction, which can be detected by urine examination (2).

Following Myerian teaching, it is advisable to fully hear the patient's version of the story, without *in toto* having to believe it, and establish when possible a reasonable rapport.

Psychotherapy, only of a superficial kind, is of some benefit. A satisfactory relationship with the psychiatrist may be the first genuine adult relationship that the patient had ever made and can be the beginning of many others that he could not have otherwise achieved. These patients are more in need of understanding than condescension. As psychotherapy proceeds a realistic appraisal of his interpretations may be possible.

Milieu therapy, correcting environmental stresses, where applicable, is useful in psychogenic paranoid reactions. Theraby on similar lines may be adopted in the chronic paranoid states as well, but is likely to be difficult.

Reassurance, practical and sensible advice, encouragement to keep delusional ideas within social bounds – all these help them to some degree to maintain a social front for years. At times a vicious circle is responsible in the perpetuation of a chronic paranoid state, and if one can succeed in breaking this, recovery or some improvement can be expected.

Anxiety is a concomitant in most patients suffering from paranoid states, and drug therapy is indicated for its symptomatic relief before any form of psychotherapy can be planned. Diazepam, 10–30 mg a day, or chlordiazepoxide, 20–40 mg a day, in divided doses is useful in allaying anxiety. In the minor forms of the illness a nightly dose of methaqualone, 150–300 mg, ensures adequate sleep. Paranoid persecutory delusions that are part of a depressive psychosis respond to a course of E.C.T. In cases where the delusions are bizarre and incomprehensible, the possibility of schizophrenia is to be borne in mind, and adequate therapy with phenothiazines and E.T.C. instituted (22). In the initial stage, where there is much agitation, violence or tendency to act-out, parenteral form of therapy with chlorpromazine, 150-200 mg, or a combination of thioproperazine 15 mg with promethazine 25-50 mg may be required. When oral medication can be relied upon–paranoid patients are very suspicious and are know to hide the tablets under the tongue and spit them out the moment the nurse leaves them-chlorpromazine in doses of 500–800 mg a day, with a larger fraction of the dose at night is helpful. Delusions and hallucinations, however, are brought under control with trifluoperazine in dose of 15–30 mg a day, combined with an antiparkinsonian agent like trihexyphenidyl 4–8 mg a day, in divided doses. Modified E.C.T. twice a week initially and as improvement is noticed at weekly intervals, upto a total of 10 to 12 may be necessary. Dosage of drugs as well as the rate at which they are reduced depend on the severity of the condition and the rate of improvement. A beneficial response can confidently be expected in these severely disturbed paranoid schizophrenics, and a socially passable behaviour achieved. With modern forms of drug therapy, combined with E.C.T. and milieu therapy the need for prolonged hospitalisation is becoming less. Continuation of chemotherapy on out-patient basis and periodic visits for psychotherapy will then be needed, may be, for several months.

In any case of paranoid disorder, once the clinical picture suggests schizophrenia, withholding active physical methods of treatment would be unpardonable. There is no harm in a therapeutic trial. A favourable response helps confirm the diagnosis of paranoid schizophrenia, since in paranoia or paranoid states results of physical methods of treatment are virtually without benefit, barring the paranoid states based on reversible disturbed brain metabolism.

Tact is needed in dealing with agitated and very suspicious patients in order to get them admitted to hospital. If possible one should avoid hospitalisation. Patients who are murderous and have made repeated assaultative attempts are dangerous and commitment becomes imperative to prevent acts of violence. Each case is to be assessed on its own merits and success of therapy depends on the experience and wisdom of the psychiatrist and his preparedness to take risks. Successful outcome is also dependent on the trust the patient develops in the psychiatrist and the tactful way in which he is managed, with aid of drugs or without. In most cases the psychiatrist should be satisfied with social recovery in his patient and any attempts at changing the personality pattern are only likely to be futile.

Psychosurgery, in the form of frontal lobotomy, may be contemplated in the paranoid schizophrenic unresponsive to a fair trial of drugs and E.C.T. provided there has been no deterioration (21).

(Common trade names of drugs mentioned : diazepam–Calmpose; chlordiazepoxide-Equibrom; methaqualone–Melsedin; chlorpromazine–Largactil; thioproperazcine–Majeptil; promethazine–Phenergan; trifluoperazine–Eskazine and trihexyphenidyl–Pacitane,)

PROGNOSIS

Deterioration may be expected in patients with more or less clear-cut schizophrenic features when the paranoid element begins to recede into the background as the illness progresses.

The prognosis in cases of paranoid schizophrenia depends on prompt and effective therapy, utilising drugs and E.C T. Relapses are likely unless a prolonged maintenance treatment is ensured after discharge from hospital. Contrary to the widespread belief every paranoid schizophrenic is not necessarily potentially homicidal.

Some cases of paranoid reactions may recover and maintain a social front and efficiency for years even though their delusions may remain, and in some instances, encapsulated. Acute exacerbations can be expected at any stage, requiring medication or short period of hospitalisation. Paranoid states with obvious precipitating factors in the environment, provided the personality of the patient remains intact, have a favourable prognosis.

Symptomatic paranoid states precipitated by acute organic illnesses, addictions or intoxications have a favourable prognosis.

Classical Kraeplinian paranoia is incurable.

CONCLUSIONS

A study of the paranoid disorders, in their protean forms, gives one the impression that there is a continuum, at one end of which there is Kraepelinian paranoia with systematised delusions and no disorganisation of the personality and at the other paranoid schizophrenia with profound personality deterioration. In between seem to appear forms of illnesses with some admixture of schizophrenic colouring.

The chronic paranoid psychoses of temporal lobe epileptics, alcoholic paranoid psychoses and amphetamine psychoses, together with other paranoid reactions occurring in chronic brain syndromes of varying pathology do strongly point to certain, still undiscovered, brain mechanisms that come into operation, either by a process of excitation or inhibition, producing paranoid disorders closely resembling the functional illnesses. The advanced age in which most paranoid manifestations are noted, irrespective of the basic pathology, is also a point in favour of specific brain mechanisms going out of gear. Furher clarification is only possible when we know more about the interconnections of the frontal and temporal lobes of the human brain. At present there is evidence to point to the intact temporal lobes being indispensable for hallucinatory phenomena, while it is not clear yet if delusional depends on intact frontal lobes.

Schizophrenics and pre-schizophrenics have a tendency to form peculiar personal concepts. These may be expressed in an equally personal style. Information flooding and ambiguity can be very disturbing to them. They allow into their thoughts irrelevant extraneous factors and their output suffers because of over-inclusion and difficulty in picking wheat from chaff. Even simple objects and situations may engender in them a meaning which is highly personal. Paranoid thinking is related to schizophrenic thinking, "lateral thinking" and "primary process" as manifest in dreams, satire and humour. They have some relation to all creative achievements. Their inter-relations would appear to the subtle and need closer examination. It may then be possible to recognise the vulnerable people at the right time and protect them from slipping into a state of unintelligibilty. Methods of improving communication between individuals at all levels and reducing distortion to the minimum also require study.

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A REVIEW OF SOME PREVENTABLE DISEASES IN THE ARMY-DECADAL PROGRESS

BY

Lt Col J.S. Lamba* and Shri K.K. Gupta**

INTRODUCTION

The Armed Forces possess a well knit, self-contained and versatile machinery to deal with various health problems among the forces and their families. To assess the magnitude of such problems in regard to their nature, size, distribution and to determine the variation in the disease pattern from time to time, health statistics play a fundamental role. Health statistics serve as yardsticks to measure the health state in its correct perspective, apply appropriate preventive and health promotive measures, evaluate the success or failure of various health programmes and for prediction of health trends. To serve as yardsticks, it is imperative that the statistical data are compiled, and analysed with the greatest possible accuracy. The pattern of diseases in any community is not constant. Every decade produces its own pattern for some diseases. In highly developed countries, communicable diseases have declined considerably and have been replaced by non-communicable and constitutional diseases. The object of this paper is to review the trend of overall morbidity and incidence of major preventable diseases in the Army in the decades 1962-71 and compare the variation of such trend during the preceeding decade 1952-61. The material has been extracted from medical statistical date held with the Army Statistical Organisation (Med) and Army Health Reports 1952-71. Though there are significant variation in the morbidity for various diseases among the Officers, JCOs/OR and NCsE, overall morbidity among all ranks has been taken for the purpose of this study.

TOTAL MORBIDITY

Total admission rates during the decade 1952-61 varied from 227.99 to 367.29 as against 201.31 to 253.41 in the last decade. There has been a progressive decrease in the incidence of total morbidity during both the decades. The downward trend is mainly attributed to a decline in the incidence of constitutional diseases. Graphical representation of year-wise total morbidity is given below :—

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Total Morbidity (Rates per 1,000) among Troops During Decades 1952-61 and 1962-71

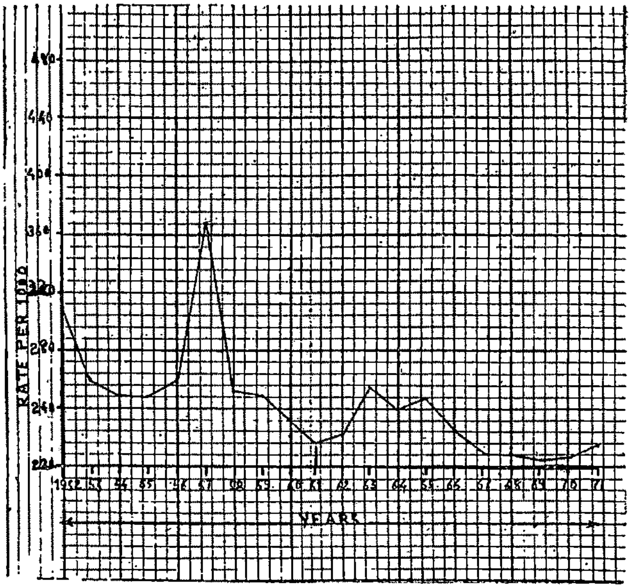

Fig. 1

MALARIA

Incidence of Malaria registered a spectacular and progressive decline upto the year 1966 after which there has been a steadily rising trend among troops as in the neighbouring civil population. National Malaria Eradication Programme was launched in 1958. The aim of this National Programme was to exterminate the disease from the country by a thorough and effective attack on the vector mosquitoes and the parasite in human reservoir over a limited period of time. It appeared the year 1966 that the aim of eradication of malaria was nearing achievement, but there has been a serious setback and the incidence of disease is steadily rising since that year Though the overall incidence of the disease does not appear to be very high, the endemicity of the disease is showing a rising trend in a large number of military stations. Graph showing annual incidence of Malaria during the two decades is given below :—

There have been increasingly larger number of outbreaks of the disease in different scattered areas in different states notably Gujrat, Rajasthan, Maharastra, Madhya Pradesh, Punjab and Haryana. Troops in military hospitals were affected, though to a much lesser extent wherever outbreaks of disease appeared amongst the neighbouring civil population. Occurence of Malaria amongst troops specially in the form of outbreaks is attributed mainly to complacency regarding the disease as anti-malaria measures are required to be observed in the Army as during the attack phase of NMEP irrespective of the stage of eradication programme in the neighbouring civil population. This complacency arose due to continued absence of the disease for a number of years in most places but has now to be curbed. Reappearance of malaria is partly attributed to appearence of resistant strain of vector mosquito to DDT or of parasite to 4-Aminoqunolines. With a view to detect possibility of vector resistance DDT susceptibility tests should be carried out periodically at affected military stations. Wherever vector resistance to the commonly used insecticide or parasite resistance to the anti-malarial drugs comes to light, other suitable substitutes must be brought into use soon and their availability in the required amounts ensured. Strategy for implementation of National Malaria Eradication Programme should be reviewed periodically to avoid malaria becoming a major public health problem again.

Malaria (Rate per 1,000) Among Troops During Decades
1952-61 and 1962-71

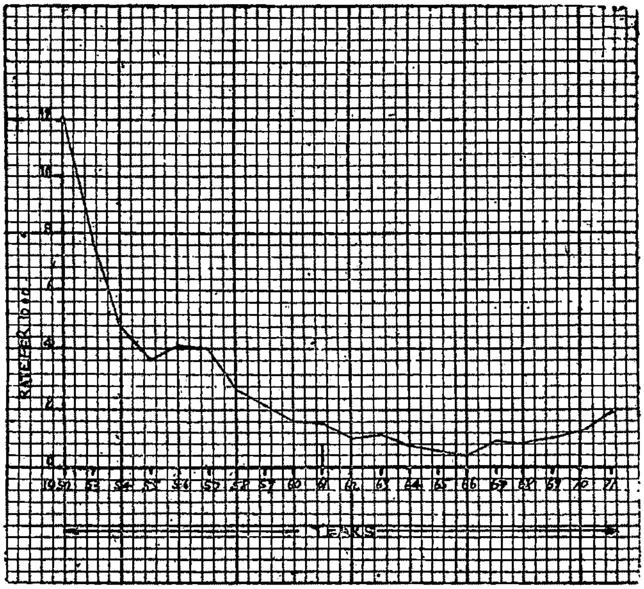

Fig. 2

INFECTIOUS HEPATITIS

This disease has also been showing a rising trend in spite of efforts to improve the enviornmental sanitation and enforcement of other control measures. Annual incidence of the disease during the last two decades is shown in the graph below :—

The incidence which stood at 3.81 per 1,000 during 1962 rose progressively to 4.59 per 1,000 during 1961 and to 5.79 per 1,000 during 1971. The peak of incidence (6 36 per 1,000) occurred in 1956 in the decade 1952-61 due to explosive water borne epidemic which started in Dec 1955 in Delhi. The peak incidence during the later decade occurred in 1966 due mainly to two water borne outbreaks, one in Eastern

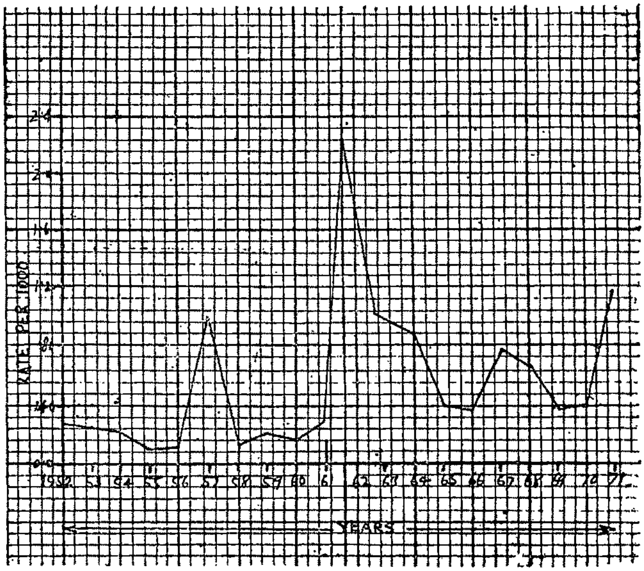

Fig. 3 a

Infectious Hepatitis (Rates per 1,000) Among Troops During Decades 1962–61 and 1952–71

Command from pollution of a Railway water supply on which the local formation and travelling trrops were dependent and another localised outbreak in the Southern Command due to pollution of a sub-soil water well. Higher incidence in between the peak periods is attributed mainly to endemic prevalance of the disease in all Commands, though sporadic localised outbreaks also contributed a sizable proporation of cases. This disease is transmitted mainly by foecal-oral route. Whereas the incidence of other excremental diseases, eg–diarrhoeal diseases and dysentery have shown a significant decline but the control measures applied for this disease have not resulted in the expected decline in morbidity so far. In the light of improved overall hygienic conditions in the army, the rising trend of morbidity due to infectious hepatitis in contrast to a progressive decline in incidence of other excremental diseases is difficult to explain. In a survey by Lamba JS (1968-69) it was found that about 70 per cent cases among troops in J&K acquired infection from outside their units by eats/drinks from insanitary places while on leave or on duty. The disease is endemic in all parts of the country and the incidence among civil population is also showing an upward trend. Due to exceretion of the virus during incubation period and 3–4 weeks after onset of jaundice, the chances for widespread dissemination of the causative agent are high. The incidence of parentrally transmitted disease is not known. Measures towards a more vigorous control on water supplies to avoid possible contamination, foolproof water purification arrangements, health education drive among troops especially in regard to their eating habits from insanitary stalls and personal hygiene should help in reducing the incidence of this disease.

DYSENTERY AND DIARRHOEAL DISEASES

The incidence of Dysentery, and diarrhoeal diseases during the last 2 decades is shown on the graphs below :—

Dysentery Including Amoebiasis (Rate per 1,000) Among Troops During Decades 1952-61 and 1962-71

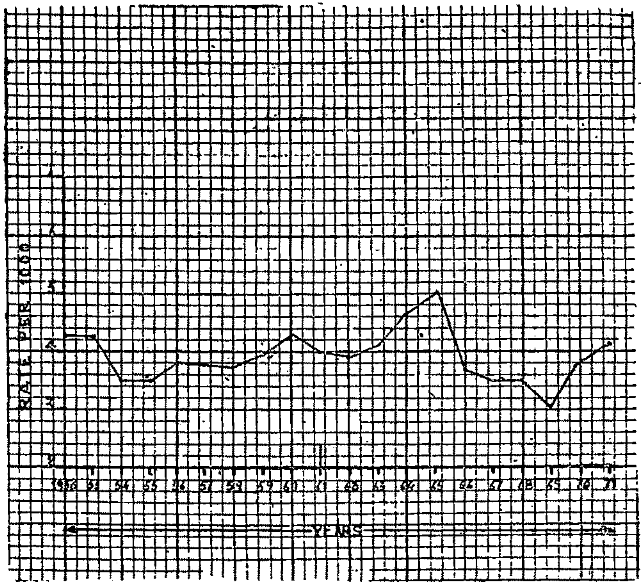

Fig. 3 b

Diarrhoeal diseases (Rates per 1.000) Among Troops during decades 1952-61 and 1962-71

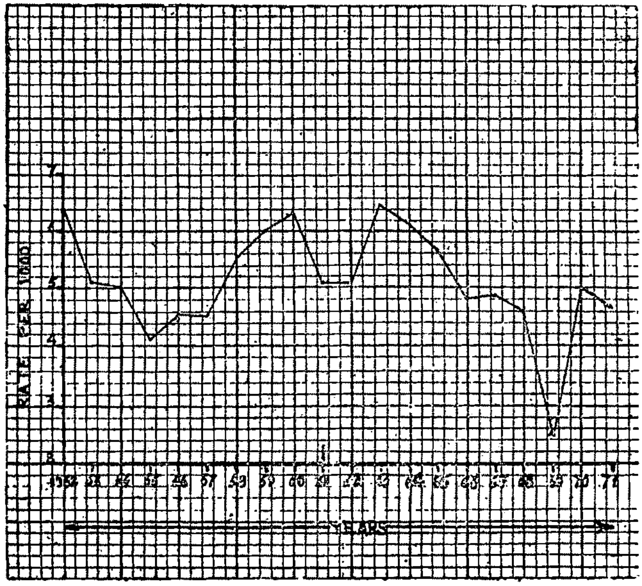

Fig. 4

The incidence of Dysentery and diarrhoeal diseases has recorded a significant decline during the later part of the decade 1962-71. This is attributed to improvements in environmental sanitation, introduction of water borne sanitation in new constructions, replacement of conservancy system in a number of old buildings, more and more fly proof kitchens and dining halls provided each year, more effective anti fly measures and health education of troops in personal hygiene.

VENEREAL DISEASES

The incidence of venereal diseases which recorded a progressive decline from 9.05 per 1,000 in 1952 to 2.41 per 1,000 in 1960 has shown a slow consistent upward trend thereafter rising to 3.95 per 1,000 during 1971. Incidence rate per 1,000 during the last two decades is shown in the graph below :—

Venereal Diseases (Rate per 1,000) among Troops during decades
1952-61 and 1968-71

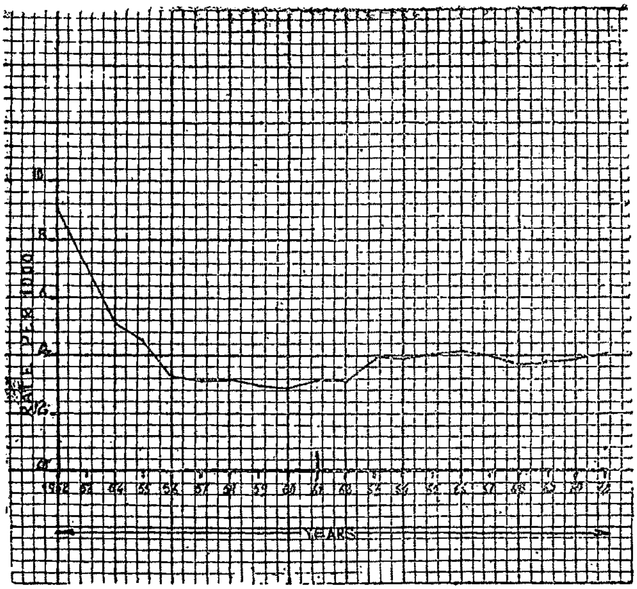

Fig. 5

The morbidity due to VD among troops usually varies in direct proportion to the incidence among civil population with whom they come in contact. Relatively higher incidence of disease among troops in the last decade is in conformity with the higher trend among the civil population during this period.

VD incidence rises usually during war time due mainly to changed psychological outlook of the soldier leading to a fall in morality standards and increase in promiscous indulgence. The rise in incidence during the Chinese aggression in 1962 and the wars with Pakistan in 1965 and 1971 was only marginal, less than 1 per 1,000 above the preceding years in comparison to 49.4 per 1,000 in 1943 from the pre-war level of 7.5 per 1,000 in 1938. This is attributed to limited sphere and duration of wars in the last decades in comparison of World War II and widespread dissemination of knowledge regarding prevention of diseases through health education programmes.

In a VD survey in J&K by Lamba JS (1968) more than 80% persons were seen to have acquired infection when they proceeded to outstations from their units on leave. A large number of exposures occurred on way to and from leave during transit near railway junctions/Military stations. This emphasises the necessity of more thorough health education of personnel proceeding on leave, providing prophylaxis material to those who desire and suppression of immoral traffic in coordination with civil authorities in important stations. As there are only a few locally acquired cases, the utility of unit prophylactic abulation rooms stressed during pre-indepence era is insignificant. Practice of abulation immediately after exposure at the place of contact should be stresed in the health education programme apart from inculcating high morality values and other preventive measures.

DIABETES MELLITUS

The incidence of Diabetes mellitus as seen from the graph below has shown a sharp rise during the last decade. The rising trend evident from mid-fiftees has not shown any tendency for decline till the end of the last decade. The reasons for a higher incidence of this disease in the army cannot be entirely attributed to increasingly greater focus of attention for screening and detction of such cases. The causes for the rise need detailed investigation.

Diabetes Mellitus (Rate per 1,000) among Troops during decades 1952-61 and 1962-71

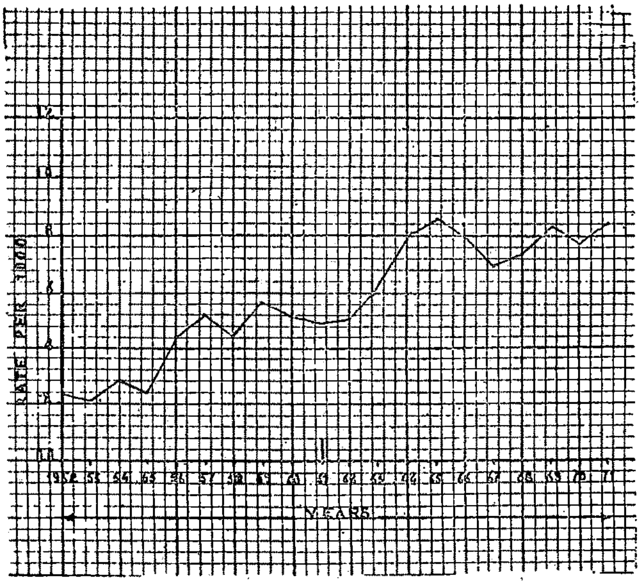

Fig. 6

ISCHAEMIC HEART DISEASE AND HYPERTENSION

The annual incidence of Ischaemic heart disease and Hypertension during the decades 1952-61 and 1962-71 is reflected in the graph below :-

Ischaemic Heart Disease (Rate per 1,000) among Troops Decades 1952-61 and 1962-71

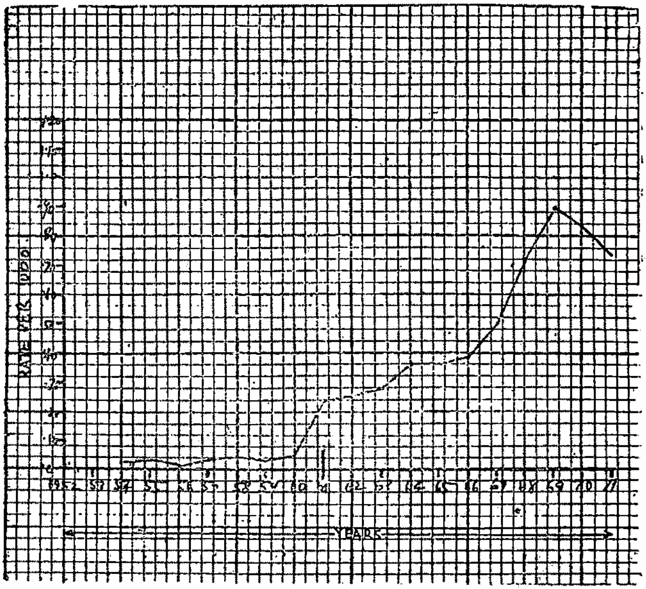

Fig. 7

Hypertension (Rate per 1,000) among Troops during Decades 1952-61 and 1962-71

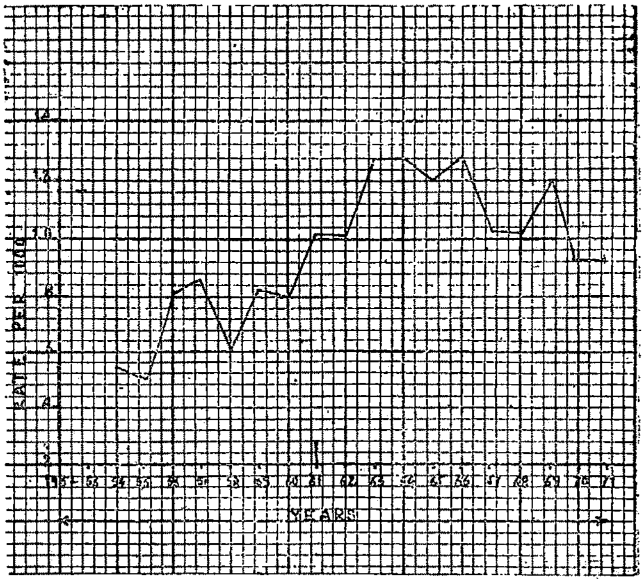

Fig. 8

The changing decadal pattern of diseases is eminently illustrated by the trend of incidence of Ischaemic heart disease which includes myocardial infarction, angina pectoris and other acute, sub-acute and chronic myocardial ischaemic conditions. The incidence rate for Ischaemic heart disease rose to 0.75 per 1,000 in 1971 (more than treble) from 0.24 per 1,000 in 1960. The incidence of hypertension, though fluctuating has also recorded a significant rise during the last decade. The rise in incidence of this group of diseases is attributed only patly to better diagnostic facilities and greater consciousness of these diseases among medical officers and the troops. The etiological factors which account for the rise of incidence among Armed Forces being multifactorial in origin cannot be easily pinpointed. With a view to curb the rising trend, it is imperative that the preventive measures are also multifactorial. Health education of troops especially in respect of more vulnerable groups towards prevention of all known factors in relation to the disease should be intensified.

PSYCHIATRIC DISEASES

The incidence of Psychiatric diseases including psychosis and Neurosis during the last two decades is shown below :-

Psychiatric Diseases (Rate per 1,000) During Decades 1952-61 and 1962-71

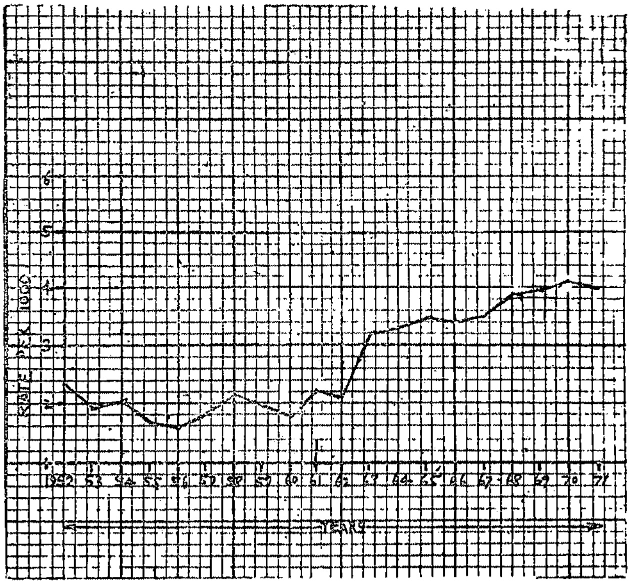

Fig. 9

Whereas there was not any significant variation of overall annual incidence of Psychiatric diseases during the decades 1952-61 but there is a substantial rising trend of these diseases during the last decade, the incidence rate rising to about double (4.01 per 1,000) in 1971 from 2.04 per in 1962. The factors responsible for the rise need investigation.

TUBERCULOSIS

Annual incidence of Tuberculosis is shown in the graph below :-

Tuberculosis (Rate per 1,000) Among Troops during decades
1952-61 and 1962-71

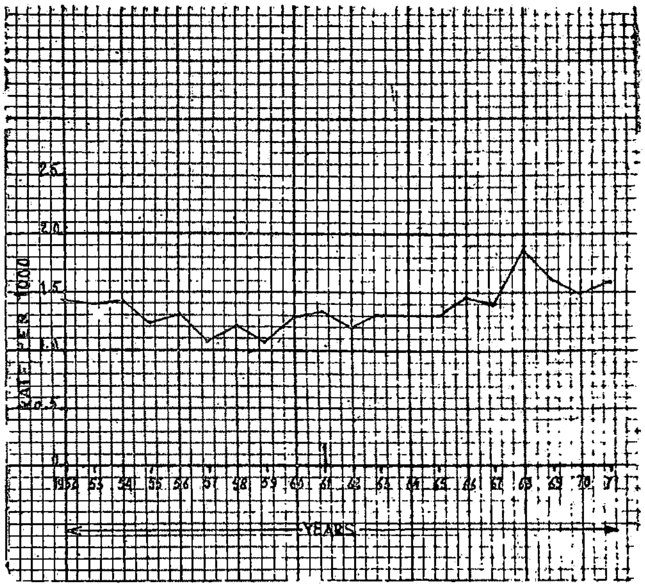

Fig. 10

It is seen from the hospital admission rates that the incidence of tuberculosis is showing a rising trend during the last decade. The incidence rose to 1.60 per 1,000 during 1972 from 1.19 per 1,000 in 1962 and 1.41 per 1,000 during 1952. Though the rise is only marginal, it is difficult to pinpoint the reasons for this upward trend. There has been consistent improvement in the living condition of troops and nutritional status is on the whole good. Recruits and young soldiers are immunised with BCG under the national tuberculosis programme. No epidemiological factor which can possibly contribute to the rising trend has come to light. Possibility of some Resurvey Medical Board Cases erroneously shown as direct admissions by some hospitals in thier morbidity data to raise the overall admission rates cannot be ruled out. Relatively higher admission rates could partly be attributed to better diagnostic facility and detection and hospitalization of casses in early stages.

SKIN DISEASES

There has not been a marked variation in morbidity due to skin diseases between the two decades. The maximum and minimum incidence during 1952-61 decade was 28.44 and 18.71 respectively whereas during the decade 1662-71 it was 24.99 and 18.97 respectively. A graph showing the comparative incidence between the two decades is shown below :—

Skin Diseases (Rate per 1,000) among Troops during decades 1952-61 and 1952-71

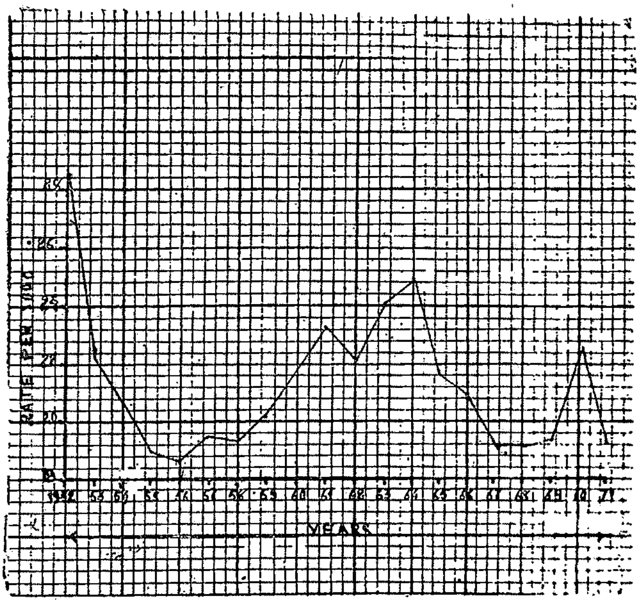

Fig. 11

RESPIRATORY INFECTIONS

The incidence of upper Respiratory infections as seen from the graph below has recorded a significant decline during the last decade :—

Respiratory Infections (Rate per 1,000) during decades 1952-61 and 1962-71

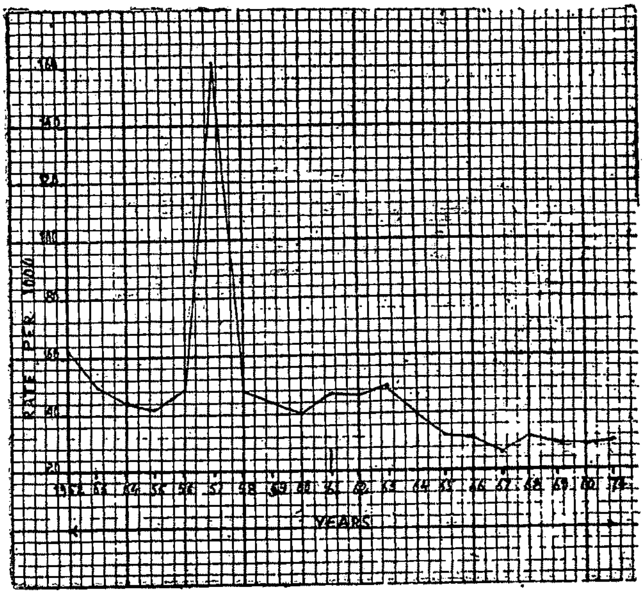

Fig. 12

A steep rise during 1957 was due to Asiatic Influenza pandemic during that year. A decline in the overall incidence of respiratory infections during the later decade is attribued to improvements in living conditions and better medical care of cases in MI Rooms reducing the number of hospitalizations.

EFFECTS OF REDUCED TEMPERATURE

Annual incidence of Effects of cold during 1952 to 71 is shown in the graph below:-

Effects of Reduced Temperature (Rate per 1,000) among Troops During Decades 1952-61 and 1962-71

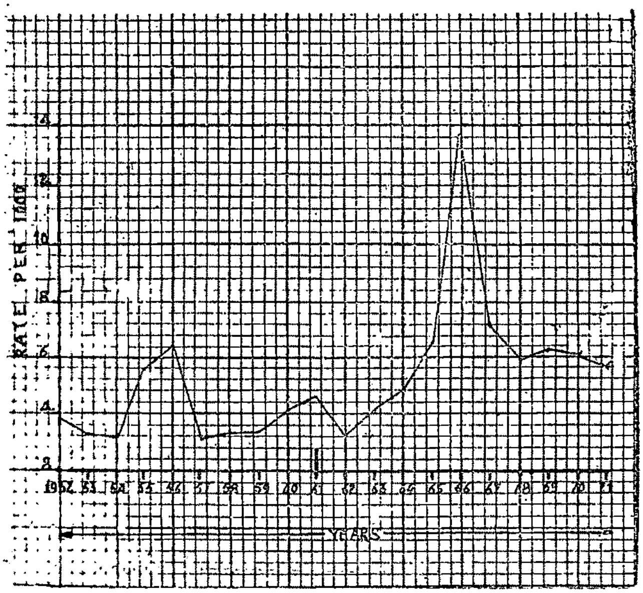

Fig. 13

Higher incidence of Effects of cold during the decade 1962-71 compared to the preceeding decade is due to a larger deployment of troops in the high altitude areas as an aftermath of Chinese aggression in 1962. The incidence showed a rise during the war with China in 1962 and during 1966 after the war with Prkistan and in 1971 during and after the war with Pakistan. The necessity for effective measures to combat effects of cold during the operations in high altitude areas to avoid manpower wastage cannot be overemphasised.

SUMMARY

Total admission ratas for overall morbidity in the Army has recorded a progressive decline during the last two decades. The downward trend is however attributed mainly to constitutional diseases. Out of the common communicable diseases, only the respiratory and intestinal diseases have contributed to the downward trend. There has been a trend towards the rise in incipence of Malaria, Infectious hepatitis, Venereal diseases and tuberculosis which are normally amenable to moderm preventive meeasures. The rising trend of these diseases is therefore a matter of concern and a pointer towards formulation of more effective preventive strategy. Non communicable diseases which are partially amenable to prevetive measures and have shown a rising trend during the last decade are Ischaemic heart diseases, hypertension, Psychiatric diseases, Diabetes mellitus and effects of reducd temperature. The rising trend of the above constitutional preventable diseases is partly due to better diagonostic facilities and greater consciousness of these diseases among medical officers and troops but it is attributed mainly to the changing pattern of diseases among the population in advancing communities. Higher incidence of morbidity due to effects of cold is evident due to a larger deployment of troops in high altitude areas after the Chinese aggression in 1962. With a view to ensure that such reviews and health reports present a realistic picture and serve as yardsticks to evalute the success or failure of various health programme, it is imperative that medical statistical data are compiled with the greatest possible accuracy.

ACKNOWLEDGEMENTS

We feel deeply grateful to Shri B Sitaraman, Director ASO for the encouragement and guidance in preparing this paper. Our thanks are also due to Mr PL Soni Asstt ASO Med for rendering assistance in of data.

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AN INVESTIGATION INTO SPECIFIC NATURE AND INCIDENCE OF SMALL BOWEL LESION IN CASES OF DYSPEPSIA IN SERVICE PERSONNEL WITH RADILOGICALLY NORMAL STOMACH DUODENUM AND COLON

BY

Col S. Banerji*

INTRODUCTION

Accuracy of radiological diagnosis of localised lesions of the small gut is virtually impossible by routine barium follow through because of the segmentation of the meal due to pyloric control over the rate of flow of the barium. Hence lesions of the small bowel are invariably missed in a large number of cases of dyspepsia whose gastro-duodenum and colon are otherwise found to be normal.

The aim of this departmental study was to investigate in detail the incidence and nature of small bowel lesions in such cases and the object was to find a simple and easy method towards their detection for correct diagnosis (where by appropriate treatment is instituted leading to a reduction in hospital attendance/bed occupied by these so called "dyspepsia" cases.

MATERIALS AND METHODS

A total of 228 cases of clinically labelled "Dyspepsia" in service personnel only, were investigated radiologically by contrast studies, during the period October 1970 to September 1973 in the Radiology Department, Armed Forces Medical College, Pune.

In most instances, the radiological investigations consisted of the following two preliminary stages of Barium studies :—

(a) First by a routine Barium meal examination for the detection of any possible causative lesion in the upper G.I. tract, namely oesophagus, stomach and duodenum.

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(b) secondly by a routine Barium enema to exclude or otherwise any lession of the large gut only if no radiological demonstrable cases of the dyspepsia was demonstrated in the above two routine examinations. the patient was subjected to a special radiological study for his small intestines, in futher search and elucidation of the cause of his dyspeptic symptoms.

The methods tried out for the radiological visualisation and examination of the small intestines were as under :-

(a) By intubation method (i.e. so called "small bowel enema").

(b) By lying the patient in the right lateral position following the ingestion of 4 ozs of the usual barium suspension in water coupled with the prior administration of Atropine 1/100 gr. I.M.I. at least 80 mts before the examination.

(c) By lying the patient in the right lateral position following the ingestion of 4 ozs of a barium mixture prepared in ice cold isotonic saline (& not water) to induce gastro-colic reflex.

The first method ("small bowel enema") besides being a complicated and cumbersome technique, and having not been consistantly successful, was given up in the very early stages of the investigation, It was so time consuming, tedious both to patient and staff and in addition, entailed more radiation in the shape of frequent preliminary fluoroscopies to determine the site of the tube tip.

Subsequently in the search of a simpler and less bothersome technique, method (b) was resorted to But this too, besides being not fully satisfactory in many cases and in others the patient complaining of some adverse side effects of the drug, was also given up. Method (c) was finally employed. It was found to produce a reasonably good diagnostic delineation of fairly large segments of the small bowel, beside having the added advantage of being the simplest and most practical method which can be easily carried out by anyone, in an otherwise busy radilogy department. The isotonic saline content of this barium mixture ensured rapid transit so that the examination could also be completed in a relatively shorter time. The drawback noted was that the mucosal coating of the intestine was not altogether satisfactory. Never the loss this was more than compensated by the case and simplicity of the techique.

With any of these methods, the preliminary preparation consisted of starving the patient for at least 12 hours prior to the examination, and allowing the previous early evening meal to be a very light one. No previous aperients were normally advocated unless warranted by a history of habitual constipation.

Following the adminstration of the Barium mixture (method 'C'), and from after about 15 to 30 mts, serial films (i.e. progress meal studies) of the filling of the small bowel segments, supplemented by local spot films of any suspected area, were carried out.

The timings of the serial films were determined and modified from case to case depending upon the rate of transit of the barium observed on constant scruting of the wet films – the aim being to obtain as such as possible, the contrast out lined image of the entire jejunal and ileal segments on these films right upto the ilco-caceal region. Whereever any obstructive element was suspected, delayed films or even 24 hours film was of necessity taken.

RESULTS

Out of the total number of 228 "Dyspepsia" cases in Service personnel only investigated, 22 cases were found positive for some lesion in the gastro-duodeunm (App. 'A'). Similary a futher 4 cases were found positive for some lesion in the large gut (App. 'B'). Thus some demonstrable cause for the patient's complaint having been found in these 26 cases (22 + 4) they were not subjected to the small bowel study.

Of the remaining 202. cases, 190 cases turned out to be thoroughly normal radiologically – in that there was no unequivocal detectable pathological findings either in the oesophagus, gastro-duodenum, colon or small bowels.

Only in 12 cases some pathology in the small bowel were found (App. 'C'). Their gastro-duodenum and colon were normal. Thus the incidence of small bowel lesions detected in cases of dyspepsia in service personnel with normal gastroduodenum and colon, constituted to 5.2% of the total series.

The above findings are presented in a tabular form.

Total No of cases of Dyspepsia in service personnel investigated radiologically.	No of cases	Percentage 100%
(a) No of cases of found positive for some lesion in the gastro-duodenum.	22	9.6% Approx.
(b) No of cases found positive for some lesion in the large bowel.	4	1.7% Approx.
(c) No of cases found positive for some lesion in the small bowel.	12	5 2% Approx.
(d) Remaining cases found thoroughly normal on barium radiological studies.	190	83.3% Approx.
Total	228	99.8% Approx.

Total of (a) + (b) + (c) = 22 + 4 + 12 = 38 (or 16.5%).

DISCUSSION & CONCLUSION

The result of the foregoing observations indicate that only in some 16.5% or cases of "Dyspepsia" in Service personnel significant lesion is demonstrable radiologically by the Barium Studies (i.e. the cause being in the G.I. tract).

But in the vast majority (83.3%) of such cases, Barium X-Ray studies are non contributory towards revealing any causative pathology, Obviously in these cases the cause must be elsewhere and it could be either organic (e.g. in other organs like gall bladder, billiary passages, pancreas, liver) or else probably functional. The latter could be psychogenic or else non-psychogenic (i.e. allergy, hypoglycaemia, drugs, reflex effects etc).

However out of the total of 16.5% positive cases, so detected after these Barium Studies, lesions of the gastroduedenum constituted the majority (9.6%), next were the lesions of the small bowel (5.2%) and lastly those of the large but (1.7%) as probable cause of the dyspepsia in the service personnel.

That barium detectable lesions of the small bowels do constitute a proportion, though small, of such cases as the cause of the dyspepsia is amply borne out by the findings of this departmental study. Hence possibility of a lesion in the small bowels should not be overlooked in the routine usually resorted to.

The results of study also indicates that in the barium investigation of such cases it will perhaps be advantageous to :—

(a) First carry out a barium meal investigation of the udper G.I. tract (oesophagus, stomach, duodenum);

(b) Failing to demonstrate a causative lesion in the upper G.I. tract, then the next step should be to explore for any possible lesion in the small bowels by the simple non cumbersome and practical method (method c) already enumerated (in fact a competent radiologist can combine the above two methods in the same sitting using the Barium Isotonic Saline suspension):

(c) Barium enema investigation should always come last in the barium studies for the investigation of dyspepsia in our service personnel.

SOME ASPECTS OF THE WOLFF--PARKINSON WHITE SYNDROME

(with illustrative cases)

BY

Col G. R. Narayanan*

The recognition of Wolff-Parkinson-White (W-P-W) or pre-excitation syndrome (Wolff, Parkinson and White, 1930), in the ECG tracing is important, because it may simulate a myocardial infarction or conceal the ECG pattern of associated heart disease. Moreover, those having this syndrome are prone to episodes of supraventricular tachycardia and the ECG pattern of this syndrome may be revealed only on subsidence of the tachycardia.

To recapitulate, the essential diagnostic features are :-

(a) Short P-R-interval of 0.10" or less.

(b) widened QRS complexes (0.11" to 0.14")

(c) slurring at the onset of the R wave, called the Delta wave.

The shortened P-R interval compensates for the widening of the QRS complexes and hence the P-J interval (from P to the end of the QRS) remains within normal limits.

Three illustrative cases are described· The first two were in candidates for commissioning in the Air Forces, whose ECGs on routine examination showed some abnormalities, superficially resembling myocardial infarction. In addition, the second case had an episode of supraventricular tachycardia, on subsidence of which the ECG pattern of W-P-W pattern was clearly discernible.

The third was in a serving officer, who cmplained of chest pain and ECG taken on account of it, showed abnormalities, wrongly construed as ischaemic changes.

Case 1

K. aged 20 years, a candidate for the air force, showed abnormal tracing in his routine ECG and hence wae referred to us. His ECG showed the typical pattern of W-P-W syndrome. In his standard 12-lead ECG tracing there is QS pattern in

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III and aVL, simulating Diaphragmatic infarction. But this pattern is due to the Delta vector of the W-P-W syndrome and not due to any ischaemia, as will be explained later.

The candidate had no histoty suggestive of infarction or attacks of tachycadia. There was no abnormal physical or radiological findinig in his cardiovascular system.

Case 2

Another candidate for the air force, S, aged 21 years was referred due to abnormal findings on the ECG done at the medical board. While being reviewd, the candidate had an episode of tachycardia. ECG tracing taken during this paroxysm showed supraventricular tachycardia.

Carotid sinus massage abolished the tachy-cardia, unmasking the ECG pattern of W-P-W syndrome. There is QS pattern in V1 and secondary ST-T changes in V5 and, all due to the W-P-W syndrome. If one misses the W-P-W syndrome, these changes may be mistaken for antero-septal infarction with lateral ischaemia.

Subsequent physical and radiological examination did not reveal any abnormality in his CVS. The candidate, however, denied past history of episodes of tachycardia.

Case 3

One of the serving officers, S, aged 31 years was initially adimtted in a field hospital with complaint of vague precodial discomfort. His ECG showed abnormalities of ST-T waves and on this basis, he was diagnosed as a case of ischaemic heart disease and after treatment, was sent on sick leave.

He reported to us on completion of sick leave for review, and his resting ECG showed typical W-P-W syndrome. The tall R wave and absence of S wave in right precordial lead V1 with ST-T changes and QS pattern in aVL are all due to the Delta wave vector orientation of the W-P-W syndrome. His previous ECG tracings were obtained for perusal and they also conformed to the above W-P-W patterns. The non-recognition of the W-P-W syndrome led to the erroneous conclusion of ischamic heart disease, mistaking the above changes due to the W-P-W syndrome as due to ischaemia. His post-exercise ECG showed slight ST-T depression which is a common feature in this syndrome. His serum enzyme levels during the initial admission were within normal limits. There were no abnormal physical or radiological finding in his CVS. He had no history of paroxysmal tachycardia. He was returned to duty with the diagnosis of W-P-W syndrome. Subsequently, he has remained asymptomatic so far.

DISCUSSION

1 to 1.5 per 1000 of asymptomatic Air Force personnel were detected to have W-P-W syndrome. (Hiss and Lamb, 1962 and Smith, 1964). It is more common in

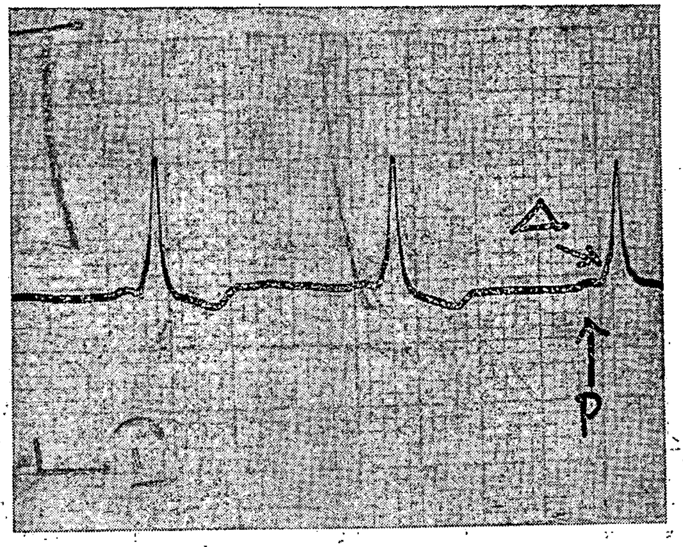
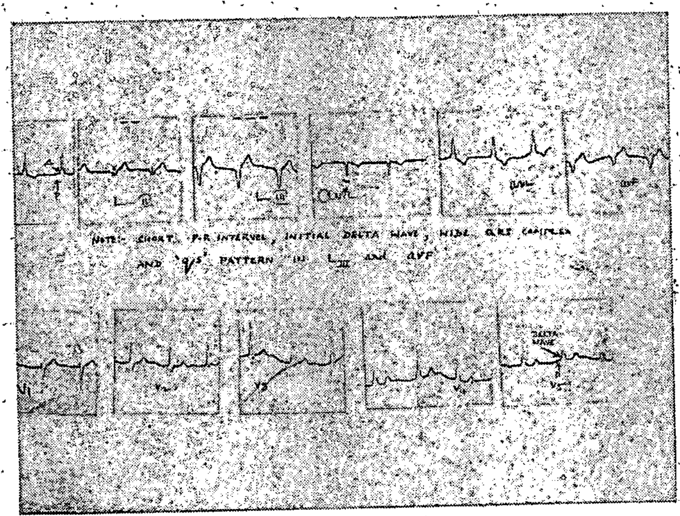


Fig. 1 Enlarged Photograph of Lead I Showing Typical Features of W–P–W Syndrome (Short P–R Interval, Initial Delta wave and Slurred wide QRS Complexes)


L II
L III
aVR
aVL
aVF
NOTE:- SHORT P-R INTERVAL, INITIAL DELTA WAVE, WIDE QRS COMPLEX
AND 'Q/S' PATTERN IN L III AND aVF
V1
V2
V3
V4
DELTA WAVE
V5

Fis. 2 Limb Leads and Precordial Leads of the same Patient Showing QS Pattern in III and aVF Simulating Diaphragmatic Infarction.

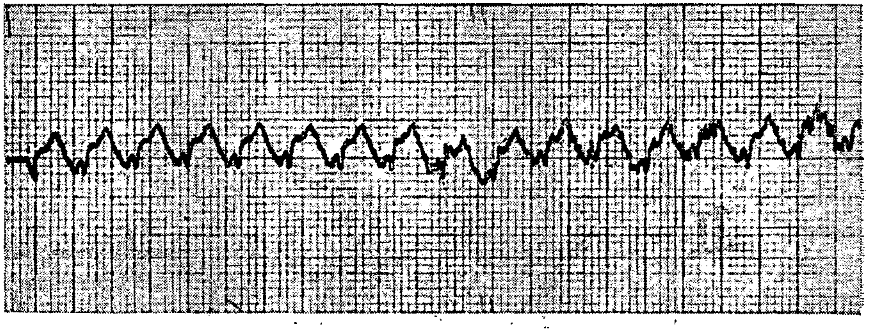


Fig. 3 Long Lead II of case No. 2, Showing Supraventricular Tachycardia.

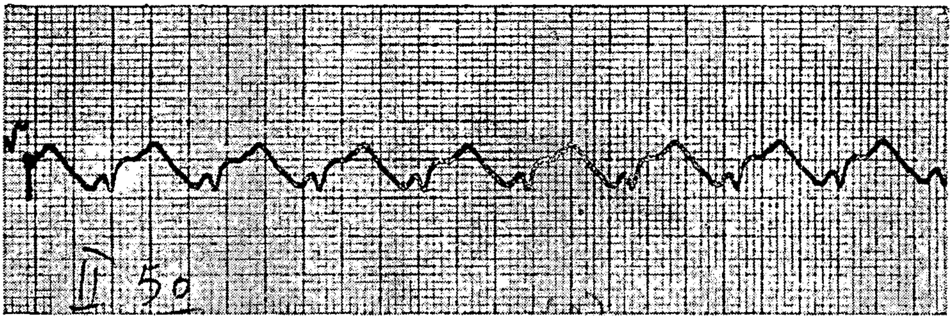


Fig. 4 Long Lead II of the same case, with Speed of 50 mm/Sec.

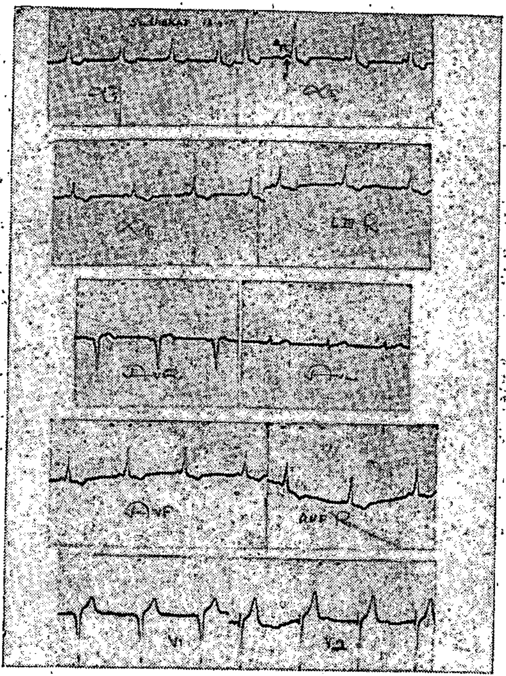
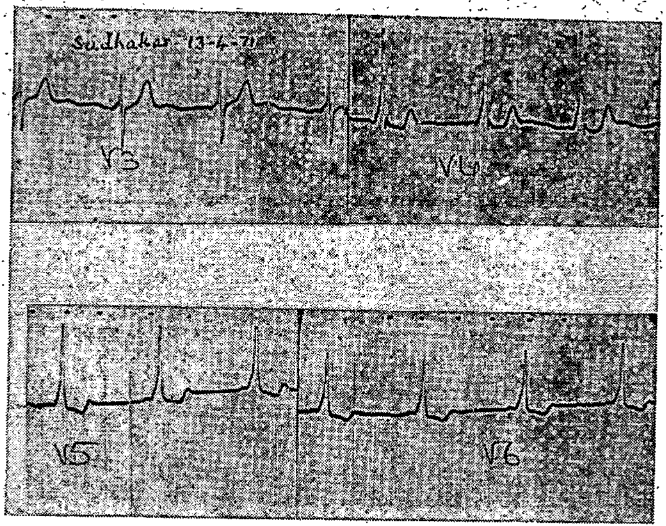


Fig. 5 & 6 Case No. 2 ECG Showing W-P-W Syndrome of type B. QS Pattern in VI and Secondary ST-T Changes in III aVF, V5 V6.



ig 6 ECG Showing W-P-W Syndrome, Secondary ST-T Changes in V5. V6.

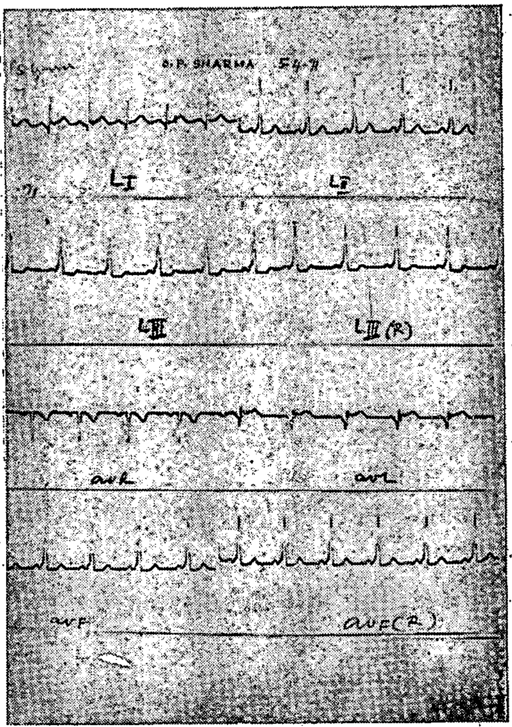


Fig. 7 & 8 ECG of case No 3, Showing type A W–P–W Syndroms with tall R in V1 and Absence of S in V1.

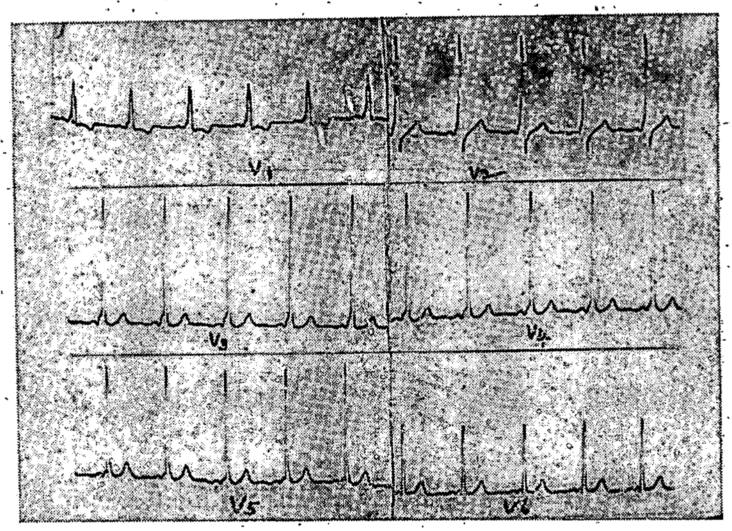


Fig. 8 ECG of case No. 3, Showing type A W–P–W Syndrome with Absence of S in V1.

males, though the Lown–Ganong–Levine syndrome (Lown, Ganong and Levine, 1952) of short P-R interval with normol QRS complexes and episodes of supraventricular tachycardia is found to be more amongst females. W-P-W syndrome may be latent and manifest at any age and may be continuous or intermittent. Amongst organic heart diseases, the congenital Ebstein's anomaly of the tricuspid valve is frequently associated with the W-P-W syndrome.

AV node at its superior and posterior margins receives fibres from the interatrial septum and Eustachian ridge which are the terminal portion of internodal tracts. In addition, a number of similar fibres enters the node laterally which may function to bypass the point of normal delay. The ECG phenomena of W-P-W complexes are commonly attributed to the aberrant conduction through lateral atrio–ventricular connections.

Short P–R interval and Delta wave has also been considered to be due to premature activation of a small region of the ventricle. Hence the condition has also got the name of pre–excitation syndrome.

There are at least three different theories for this pre-excitation :—

(a) Anamalous condition from the atria to the ventricles by way of the bundles of Kent (Mahaim, 1947 and Wolferth and Wood, 1941)

(b) focus of increased irritability in the ventricles which is caused to undergo premature excitation, perhaps by the mechanical jarring of atrial contraction, that is, in the nature of a fusion beat.

(c) certain fibres of the A–V node are damaged and there is conduction through this part without the nodal delay (Prinzmetal, 1952).

The abnormal QRS complex with its delta wave results from activation of ventricles by two wave fronts descending in parallel fashion. In this sense, the QRS is a fusion complex. Variations in velocity of conduction in these two pathways can occur due to influence of sympathetic, parasympathetic and other factors. Hence, an individual with anamalous conduction may have wide variations of QRS complexes from beat to beat or in the ECGs taken on different occasions.

The nodal defect acts as an ectopic focus, giving rise to supraventricular arrhythmias, usually atrial tachycardia but also atrial flutter and fibrillation. Occurrence of ventricular fibrillation in W-P-W syndrome has been reported by Dreifus and Reitman, 1971.

The Delta wave indicates intramyocardial spread rather than through conduction pathway and hence it is of small amplitude and slow slope. (Grant, 1957) It is usually of about 5 mm. amplitude and occurs about 0.04" to 0.06" before onset of QRS complex.

Depending on the direction of the vector of the Delta wave, the W-P-W syndrome is grouped into A and B types.

In type A, the Delta wave is directed anteriorly and either superiorly or inferiorly. In our case No. 1 the ECG shows Q S pattern in leads III and aVF, resembling diaphragmatic infarction this pattern is due to the initial Delta wave being directed anteriorly as well as superiorly. This case belongs to type A. Because the vector is pointing superiorly and away from the left foot electrode, a negative deflection of Q S is produced in aVF and III. The ST-T changes in I and aVL are secondary to the abnormal Q RS complexes in these leads and are not primary ischaemic changes. The candidate had no underlying organic disorder.

The second case belongs to type B. A Q S pattern is seen in V1 ; the Delta wave in type B is directed initially to the left and posteriorly accounting for the absence of the initial septal vector, resulting in Q S pattern in V1. This pattern, along with the secondary ST-T changes in V5 and V6 may be mistaken for anteroseptal infaraction with lateral ischaemia, if one misses recognition of the type B W-P W syndrome.

The third case belongs to type A. Here also the initial Delta vector is directed anteriorly, thus accounting for the tall R wave and absence of S wave in the right precordial lead of V1. However, the vector, apart from being directed anteriorly is also directed *inferiorly* (as in contrast to the first case, where the vector was directed anteriorly and *superiorly*). Hence, in this patient, there are positive R complexes in III and aVF also in addition to the tall R in V1. There is Q S pattern in aVL, due to the inferior direction of the vector as mentioned above. The non - recognition of this type A W-P-W syndrome had led to the erroneous diagnosis of Ischaemic heart disease. The tall R with absence of S wave in the right precordial lead V1 may also be mistaken for right ventricular hypertrophy. Parashar (1971) has reported a case of misdiagnosed intermittent W-P-W syndrome belonging to group B. The intermittent nature of the changes was another pitfall in the diagnosis, with spontaneous variations in the ECG pattern, leading to an erroneous diagnosis of ischaemic heart disease.

It is significant to point out that exercise ECG in the presence of W-P-W pattern shows invariably ST-T depression and thus does not help to rule out associated Ischaemic Heart Disease. Actually this test is not valid in the face of a baseline abnormal ECG. These ST-T changes are probably secondary, as seen in Bundle branch block (BBB). This is borne in case No. 3 where his post exercise ECG showed ST-T depression. It has been found that in intermittent W-P-W syndrome, exercise ECG shows no deterioration from the resting pattern during this period of normal conduction unless the subject has associated Ischaemic Heart Disease.

However, apart from simulating myocardial infarction, the Q RS of the W-P-W syndrome may mask the Q waves associated with myocardial infarction. In such cases, apart from the history, evolution the course of the clinical and ECG pattern, and serum enzyme studies in the acute phase, Vectorcardiogram can help. In uncomplicated W-P-W syndrome, Vectorcardiogram shows close spacing of the initial portion only. When associated with ischaemic heart disease, the vectorcardiogram shows abnormal distortions of the loop and an abnormal ventricular gradient. Vectorcardiogram is also useful in differentiating W-P-W syndrome from bundle branch block and ventricular hypertrophy, in certain doubtful cases.

CONCLUSION

Three cases of W-P-W syndrome, two of type A and one of type B have been described with analysis of their ECG pattern. While the first two cases were in candidates for commissioning in the Air Force, the third was detected in a serving officer, erroneously diagnosed as ischaemic heart disease. Awareness and recognition of this syndrome will prevent wrong diagnosis of ischaemic heart disease, bundle branch block or ventricular hypertrophy and even nodal rhythm. It is also mandatory to exclude this condition from every case showing paroxysmal supraventricular tachycardia. Last but not least. one should also be aware that this syndrome can be associated with organic heart disease, the ECG patterns of which may be masked by this syndrome. Hence, proper evaluation, including recourse to spatial vectorcardiogram if necessary, is required in such cases.

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MARCUS GUNN SYNKINESIS

(Case Reports)

BY

Major K.A. Ahmed*

INTRODUCTION

The Marcus Gunn Synkinesis is otherwise known as Jaw winking phenomenon. Marcus Gunn first described this phenomenon in 1895. Later Cooper (1937), Spaeth (1947), Warten-Berg (1948), and Simpon (1956) have also made comprehensive study on the subject. Simpson DG in Arch Ophthalmology (56.743 : 1956) pointed out that Jaw Winking is an incorrect name because it is not a wink but a lid retraction. Falls et al (1957) published review of 115 reported cases. Dhir and Agrawal (1961) reported a case following ptosis surgery.

REPORTS

Cases I & II.

The figures I, II, III and IV, V and VI are those of two daughters of a civilian clerk of Military Hospital aged 12 and 9 years respectively reported to Eye Centre with complaints of drooping of eye lids since childhood. There was no history of similar disability to any other members of the family. On examination besides local ocular abnormality no other abnormality was noted. Case I (Fig I, II, III) – There was moderate degree of ptosis in left eye seen. No other extra ocular or intra-ocular abnormality noted. Visual acuity Rt 6/6 Lt 6/6. Binocular vision – good. No paresis of any other ocular muscles noted. The ptosis was seen to be corrected on opening the mouth and deviating to the opposite direction, but not by simply opening the mouth.

Case II (Figures IV, V, VI).

The ptosis in the right eye is minimal. In this case also besides the ptosis in Rt eye no other abnormality seen. The retraction of the Rt eye lid is not very prominent. Though there is some degree of correction of ptosis is present by opening the mouth, but the normal eye i.e. the Lt eye showed some retraction of lid making the eye more prominent.

Case III (Figures VII, VIII, IX and X).

Son of a Air Force Corporal aged 5 years brought to eye Centre by his parent with the complaints of that there is drooping of the Lt Eye upper lid weich keeps on

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Graded Specialist (Ophthalmology) 150 General Hospital, C/o 56 APO

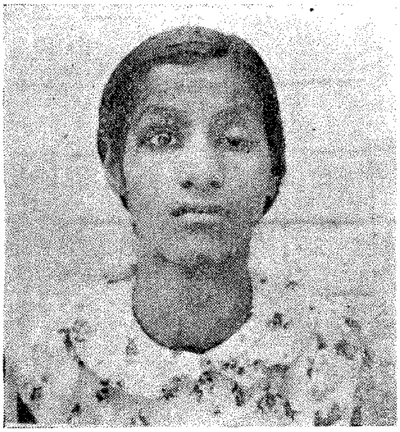


Fig. 1

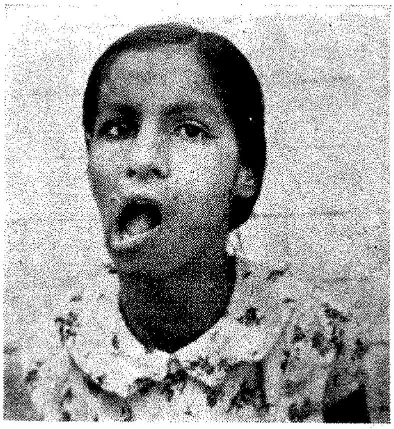


Fig. 2

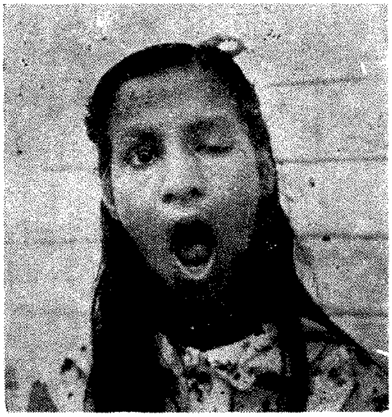
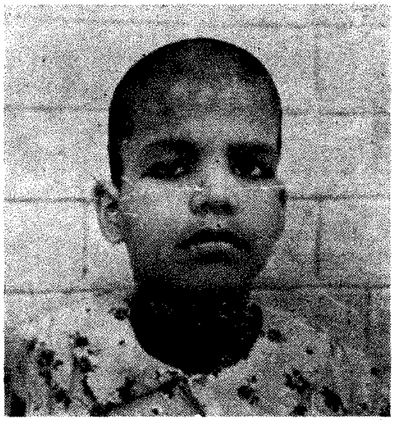


Fig. 3



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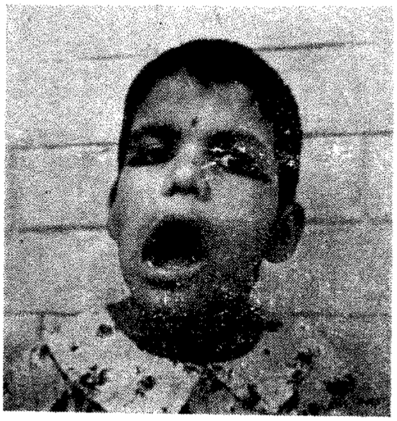

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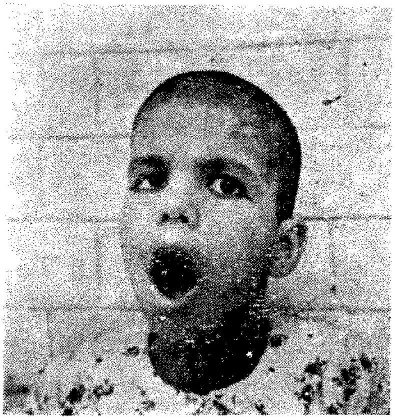

Fig. 6

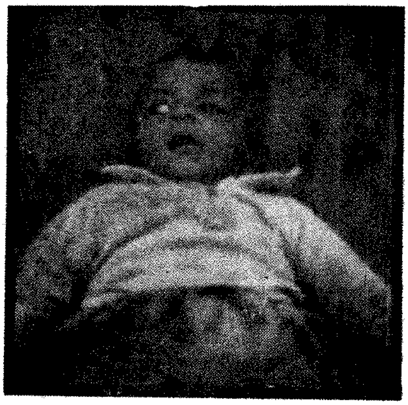

Fig. 7

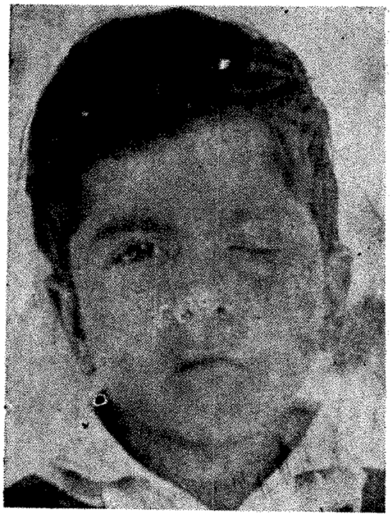

Fig. 8

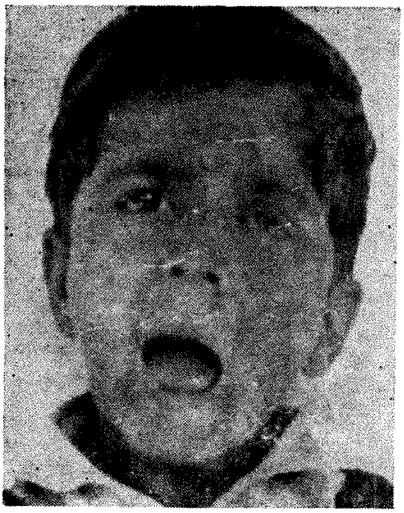

Fig. 9

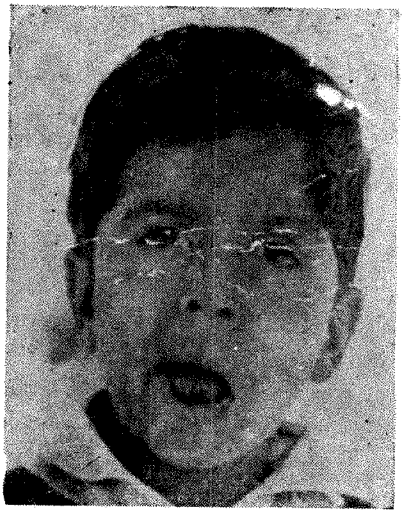

Fig. 10

unusually moving up and down during taking food or movement of mouth. The drooping of eye lid was noticed since childhood as shown in Fig. VII when the child was few months old. The retraction of eye lid of the same eye was noticed since the child started taking solid food. No history of similar disability to any other members of his family. On examination besides the drooping of the Lt eye upper lid which is being corrected by opening the mouth no other abnoamality noted in the eyes.

DISCUSSION

Marcus Gunn first described in a case—15 years old girl—having Lt sided Jaw winking phenomenon associated with inequality in size of pupil—Lt being smaller. There was also weakness of Lt orbicularis oculi. Ptosis is congenital in 90% of the cases. It may also be acquired specially in old individual. This Jaw winking is seen in 7% of the cases having no ptosis and 3% having bilateral ptosis.

This lid retraction phenomenon has been observed on following movements :-

(a) Movements of the lips.
(b) Whistling
(c) Clenching of teeth.
(d) Puffing out of cheeks.
(e) Protrusion of tongue.
(f) Swallowing.
(g) Reading.
(h) Eating.

There are at times other associated congenital abnormality eg.

(i) Ectodactyly.
(ii) Bilateral pes Cavus.
(iii) Cryptorchism etc.

There are various theories to explain the phenomenon out of which the common ones are :—

(i) Abnormal nervous connection : Some fibres of the III cranial nerve arise from the V cranial nerves nucleus and some fibres of V nerve may reach levator palpebrae via the auriculo-temporal branch.

(ii) Fnnctional Interference :- Explanation was given on physiological basis and postulated that it may be due to :-

(a) Irradiation of normal dormant connection.
(b) Release of inbibition.
(c) Spread in impulse by irradiation.

(iii) Atavistic reversion :- In fish there is strong association of Jaw opening and eye opening ie. deep muscles contracting while spurficial muscale are relaxing.

Thus a weak levator may only elevate the lid when the orbicularis is released by Jaw opening. This explanation is not also satisfactory as reverse marcus Gunn phenomenon is also seen to occur.

TREATMENT

No treatment is satisfactory. In these types of cases when ptosis is very marked and there is no paresis of superior rectus, tenotomy of the levator followed by transplantation of one third of the tendon of superior rectus may be recommended.

ACKNOWLEDGEMENT

I acknowledge with thanks and gratitude for the guidance and encouragement I had from Col GS Sihota, Adviser in Ophthalmology Army Hospital Delhi and Lt Col AK Sen, Commanding Officer 150 GH in publishing these case reports.

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A CASE OF ACQUIRED AFIBRINOGENAEMIA

BY

Major V.C. Chaturvedi*

Case Report :

SK – A young primi gravida was admitted with labour pains to this hospital. The patient was attending Ante Natal Clinic regularly and the period of gestation was totally uneventful.

The membranes ruptured at 1100 hrs and the patient delivered a male baby normally and spontaneously at 1158 hrs. The placenta and membranes were out a. 1210 hrs. The uterus was well contracted. Episiotomy was done.

It was noticed that even after about 10–15 minutes of expulsion of after–birth and though the uterus was apparently well contracted, the patient was having a constant trickle of blood per vaginum. The vagina and cervix were examined per speculum and the cervix was found to be torn laterally on left side for about one inch. The tear was caught by ring forceps and left till such time preparation for cervical repair were complete. The cervical tear was repaired by catgut at 1400 hrs. The uterine cavity was explored digitally for any left over piece of placenta or membranes, and none was found except that there was noticeable blood still oozing from the OS.

The blood was taken from a cubital vein and sent for grouping and cross matching.

After some time it was noticed that not only the vaginal bleeding has not stopped but the injection sites are also bleeding. The uterus was still contracted.

The most dangerous complication of Afibrinogenaemia flashed in the mind and blood was collected for Weiner's bed-side clotting test. BT/CT was immediately ordered and blood transfusion started till such time plasma was being made ready.

The Weiner's Test showed complete failure to clot in 10 mts. The clotting time was prolonged to more than 15 mts. The diagnosis of Afibrinogenaemia was sadly accepted to be confirmed. Double strength plasma was being dissolved. The blood and plasma bottles were made to run simultaneously, but the patient went down hill and expired at 1830 hrs. within 7 hours of delivery.

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*Gynaicologist, Military Hospital, Pathankot.

DISCUSSION

In 1901, Delee[3] described a haemophilia like phenomenon which caused death due to PPH. Dieckman[4] in 1936 for the first time suggested the possibility of fibrinogen deficiency in uncontrollable PPH. It was in 1949 that Maloney et al[7] treated a case successfully by intravenous injection of fibrinogen and proved the deficiency of the substance in such cases beyond doubt. Since then many workers have shown deficiency of fibrinogen as responsible for uterine haemorrhage. (Scot) – (II) Sargent et al[10] Lazer[6].

Mechanism of Blood Clotting :

It will not be out of place here to review our knowledge of blood clotting mechanism. There are three distinct steps in the process of blood clotting. Bourne & Willian[2].

(1) Formation of Thromobo-plastin (2) Formation of Thrombin and

(3) Formation of fibrin–These actions can be represented as follows :—

(i) Platelet factor + thromboplastinogen factor = Thromboplastin

(ii) Thromboplastin + Calcium + Labile factor (factor V) + Stable factor (factor VII) = Thrombin

(iii) Thrombin + fibrinogen = Fibrin.

In step I thromboplastinogen factor is a combination of serveral substances found in plasma. Of these, two substances are important, factor VIII, or anti-haemophilic gloubulin factor and plasma prothrombin component, PTC, or factor IX.

Normal blood fibrinogen level is 325-400 mg per 100 ml. It is a protein of high molecular weight and is synthesized in liver. Placenta, decidua and Liquor Amnii are other sources of thromboplastin. (BHATTACHARYA et al) (I)

Another form of thromboplastin, the tissue thromboplastin is present in the body, though absent from the blood.

Mechanism of Production of Defect in Clotting

The thromboplastin converts fibrinogen of the blood in to fibrin. The thromboplastin may be received from two sources either it is liberated due to premature separation of placenta or by an embolus of Liquor Amnii which contains a lot of thromboplastinogen. The mechanism can be summarised as follows :

(a) Thromboplastin acts on blood fibrinogen producing Fibrin.

Fibrin as multiple small emboli produces

- (i) Focal Necrosis
- (ii) Sudden death due to
  - (i) Amniotic embolism
  - (ii) Obstetric shock

(b) Production of enzyme fibrinlysin, Liquour amnii is rich in a fibrinolysin activator which can set in motion (when it enters circulation) the plasminogen system leading to fibrinogen depletion or failure of coagulation, causing Haemorrhage leading to Death

This depletion of fibrinogen can be caused by any of the following causes :–

(i) Accidental Haemorrhage
(ii) Liquor Amnii Embolism
(iii) Missed abortion
(iv) Infusion of dextravan or other poly–vinyl pyruolidine substances which are macro–molecular. These tend to precipitate the available fibrinogen as fibrin.

DIAGNONSIS

Persisent PPH in an otherwise contracted uterus should give suspicion of fibrinogen depletion. Quickest test, which can be done in the labour room/ward/home is Weiner's[12] test ÷ 5–10 ml blood drawn and kept in a sterile tube and observed. Normally, clotting should occur within 10 minutes. Absence of clotting within this time indicates a serious deficiency, a soft clot which dissolves readily, wholly or partially indicates not so serious a deficiency. Nevertheless it is suggestive of excessive plasminogen activity. The ciotting time is increased; bleeding time may or may not be prolonged.

Platelet count and prothrombin time may be of use. Heat coagulation test of plasma is another simple test but is not reliable (KRISHNA MENON) – [5]

Quantitative estimation of fibrinogen may be done by SHEA'S method. Accurate estimate can also be done by BIURET method (MUKHERJEE)[8] using a Bosch and Lomb spectronic 20, using a standard calibration curve. But all these methods require time, and a patient of hypo fibrinogenaemia will not give time to do all these tests.

TREATMENT

The treatment is by replacement cf fibrinogen. It may be done by dissolving fibrinogen in distilled water and giving by IV injection. The drug can be given up to 4–6 grams in 2% solution. The drug is expensive, difficult to procure, does not dissolve easily and may cause viral hepatitis. When fibrinogen is not available blood transfusion may be resorted to. Whole blood contains about 1.4 gms of fibrinogen per pint. Total requirement of fibrinogen is about 8 gm. Thus, blood is a poor substitute for fibrinogen. Quadruple strength plasma will contain up to 4.4. grams/per pint and is ideal to give. Plasma in quadruple strength has got its own problems. It is difficult to dissolve a bottle of dried plasma in one fourth of the quantity of distilled water, normally used for reconstitution. The needle blocks frequently. unless plasma is made from small pool. which is not possible to know in stored plasma, chances of infective hepatits are more and therefore the bleeding episode may be controlled but the renal and hepatic failure which may later develop may kill the patient ultimately. Blood transfusion, therefore though limited value for fibrinogen replacement, has its own advantages. It replaces blood for blood and the oxygen carrying capacity of the blood is maintained. It replanishes platelet dificienty and replaces other unrecongnised factors if any.

Macro-molecular substances like Poly-vinyl pyrrolidine-Dextravan, are absolutely contra indicated. They are harmfull in three ways. They increase the blood volume, thus diluting any fibrinogen available. They precipitate the available fibrinogen and they tend to interfere with correct determination of blood group.

PROGNOSIS

Once Hypofibirinogenaemia, is diagnosed the prognosis is always grave. Out of 7 cases seen by the author only one survived. One more case survived the episode but died ultimately, after 5 days due to renal failure and uraemia.

SUMMARY

(1) A case of fribrinogenaemia is descrided.

(2) The mechanisms of blood coagulation and its failure are discussed.

(3) Investigation, treatment and prognosis of Fibrinogenaemia are discussed.

ACKNOWLEGEMENT

I express my thanks to OC MH Pathankot, Colonel PK Basu Roy for his kind permission to publish the case.

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A CASE OF MASSIVE GIANT FIBROADENOMA BREAST WITH OBESITY

BY

Lt Col N.S. Bamrah * & Lt Col S.N. Khandekar **

INTRODUCTION

This rare tumour was first described by Johannes Muller (1838) and gave it the name of cystosarcoma Phyllodes because it contained cysts, was fleshy and presented clefts like the leaves of a book (Phyllon, leaf). Stewart (1950) has listed forty four synonyms. Few of these need mentioning such as serocystic disease of breast, giant intracanalicular myxoma and giant intracanalicular fibroadenoma. It is non-malignant inspite of term sarcoma which in days of Muller meant flesh, though it has been divided into benign and malignant variants by Treves and Sunderland (1951). This tumour makes 2.5% of all fibro epithelial tumours of the breast and 0.3% of all mammary neoplasms (Lester and Stout, 1954).

Obesity is an hazard for surgical approach, wound healing and it taxes the skill of anaesthetist to the extreme.

This case of actually giant sized rare tumour of giant fibroadenoma of breast in a grossly obese famale has prompted, us to publish it.

CASE REPORT

Female 38 years old, hieght 157 cm (5'-2") weight 94 Kgs (207 Lbs) had lump in her right breast since two years which started increasing in size rapidly since two months and attained enormous size. There was slight pain in the breast but her chief complaint was dragging sensation due to the weight of the tumour: She was mother of two children and the last child was one year old. Mass did not increase much in size during pregnancy. Right breast was grossly enlarged with stretched out skin on lateral side , and she was bent to the right due to the weight of the mass. Nipple was normal and no discharge came out on squzeezing. There were dilated veins visible under the skin over the lump which virtually occupied the whole breast and breast tissue could be felt with difficulty under and around the areola. Neither the skin was adherant to the lump nor the lump was adherant to the pectoralis major muscle. It was bosselated mostly firm but with cystic feel at places. No lymphnode were palpable in right axilla, supraclavicular region, or any-where else. X-Ray chest was normal. She had poor cardio-respiratory reserve, sebrasez test was hardly fifteen seconds even after repeated attempts. She became dyspnoeic with mild exertion and got mildly cyanosed after bout of cough. She was intubated under local analgesia, and anaesthetised only after patent airway was ensured. Simple mastectomy was carried out. Post-operative course was uneventful.

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* Classified Specialiest (Surgery) MH, YOL (KV)

** Classified Specialist (Anaesthesia) MH, JULLUNDUR.

Tumor was 30 cm x 26.5 cm (Fig. 1), it weighed 5.350 kgs. It was ovoid, well encapsulated, with slight lobulation. Cut surface buldged (Fig. 2), showed areas of fibrous tissue seperated by cysts and gelataneous mass. Typical leaf like projections of the tumour tissue into the cystic cavities were also seen. Histopathology confirmed it to be giant fibroadenoma without any evidence of malignancy.

DISCUSSION

The age of our patients 38 years more or less conformed to generally mentioned age incidence in fourth and fifth decade, though recently its high incidence in adolescent female has been reported from Japan (Minami and Masudua; 1968), from America (Amerson, 1970). Size of this tumour has been 30 cm x 26.5 cm, bigger than the largest tumour reported by Gupta and Gupta (1973) in their series of cases. Rapidity of growth has been linked with the occurrence of pregnancy in the case of Cumin (1827) and in those of Reed and Heibert (1942). In this case the tumour started growing rapidly 8 months after the delivery, when suckling was stopped. Trauma has also been suggested to be another factor connected with rapid growth (Lee and pack, 1931). It may be pointed out that skin ulceration or fungation is because of pressure necrosis and overstretching of skin and not due to the invasion. Malignant change is extremely rare and when ever it occurs, it is mesenchymatous element which undergoes such a change and epithetial element never metastise.

Treatment generally accepted is simple mastectomy. Local excision has been suggested by Wulsin (1960) but Rix et al (1971) has shown high rate of recurrence following local excision. Better results have been reported following radical mastectomy (Treves and Sunderland, 1951). As suggested by Gupta and Gupta (1973) also adequate simple mastectomy with or without skin graft is the treament of choice. Radical mastectomy should be reserved only for those when the pre-operative diagnosis is clashing with carcinama breast, or if malignancy is suspected.

ANAESTHTIC PROBLEMS OF THE CASE

The main anaesthtic problem in this case was gross obesity with poor cardio respiratory reserve. Breath holding test was less than 15 seconds. This poor reserve, caused by gross restriction of diaphragmatic movements due to increased deposition of intra abdominal fat was further reduced because of extra weight, the chest had to lift.

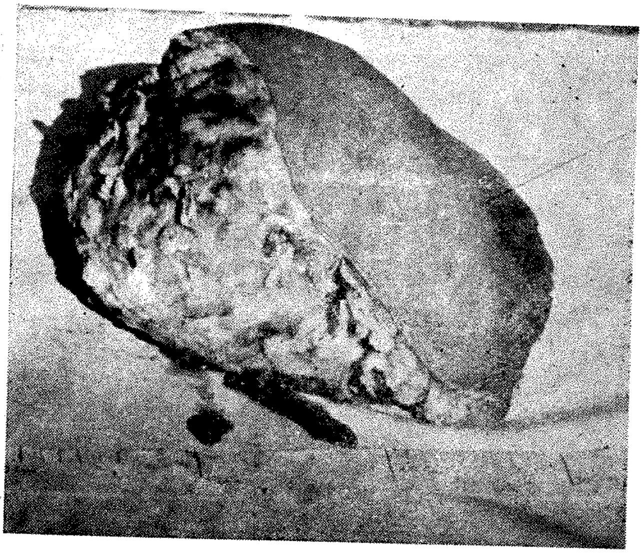

Fig. 1 Tumour 30 x 26.5 cm. Note the normal nipple and little breast tissue under the areola.

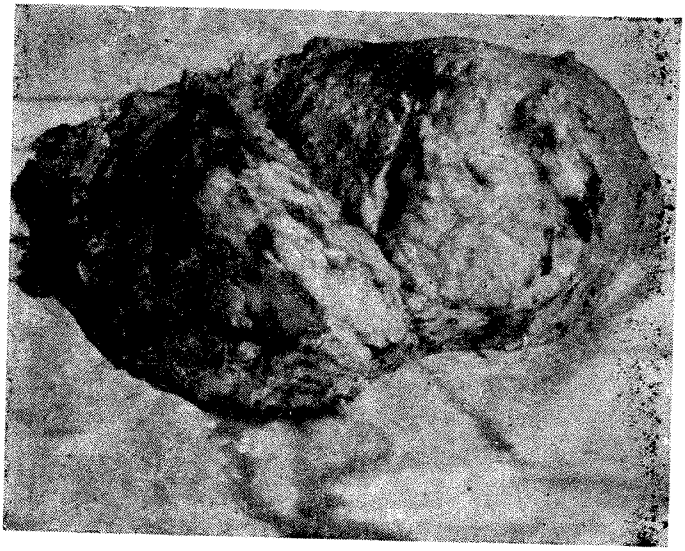

Fig. 2 Cut surface specimen of Fig. I. Note well formed capsule, fleshy mass with cystic cavities and leaf like projections.

The tumour removed during operation was 5.350 kgs. Other breast was also relatively quite big in size. It was estimated that she laboured under the disadvantage of having to lift a net weight of about 8 kg over her chest alone while breathing in supine position. Difficulty was visualised in lung inflation after administration of Suxamethonium prior to endotracheal intubation, due to increased resistance and indirect laryngoscopy due to presence of a short 'bull-neck'. Intubation, under local analgesia with patient breathing spontaneously though cumber-some was consideredsafer. It may be recalled that heaviest indvidual reported to have been anaesthetised and operated upon, weighed 454 Lbs. He got dyspnoeic in supineposition and could sleep only while sitting. He had to be intubated under local analgesia (Courington and Little Jr, 1968). An alternative could be, denitrogenation and intubation under hundred percent oxygen after thiopentons-suxamethonium sequnence. But hypercarbia was a definite possibility in event of difficulty in intubation and in case of further embarrassment of respiration due to inflation of stomach following use of higher inflation pressure before intubation.

Patient was intubated under local analgesia and anaesthetised only after patent airway was ensured, thus circumventing the above problem. A large inflationary pressure was required to over come the additional resistance inspite of curarisation. The lung inflationary pattern was therefore that of large volumes at slow rates, but this did not have any deleterious effect on the circulation. There was no post operative complication.

SUMMARY

A case of massive Giant—fibroadenoma of breast in an obese female, is reported. Short review of literature related to the tumour and its treatment is given, anaesthetic problems encountered are also given.

ACKNOWLEDGEMENT

We are thankful to LT COL RK CHOPRA, VSM Commanding MH Yol for permitting us to publish the case.

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MR-2868 Surg Lt NJ Advani has been awarded Silver Medal of the Chief of the Air Staff for standing first in the Primary Aviation Medichine Course.

MR-2865 Surg Lt VK Puri has been awarded Silver Medal of the Chif of the Air Staff for standing first in the post-graduate diploma examination in Aviation Medicine.

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3	MS-9025	Surg Lt (Mrs) Monica Mohindra	15-2-73
4	MR-1668	Lt Col JL Sengupta	15-2-73
5	MR-267	Brig S Gopalakrishnan	22-2-73
6	MR-675	Lt Col PK Chakrabarty	27-2-73
7	DR-10048 F	Lt Col BS Kochar	5-3-73
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9	MR-307	Brig KC Dasgupta, MC	11-3-73
10	MR-522	Col KSV Krishnamurthy	12-3-73
11	MR-173	Lt Gen BL Kapoor, PVSM, AVSM	14-3-73
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2	NR-12035	Lt Col (Miss) FJ Sahayar	Vishist Seva Medal
3	NR-12011	Lt Col (Miss) Y Vijayamma	'Florence Nightingale Medal' by the International Committee of the Red Cross, Geneva
4	NR-6808717	Lt Col (Miss) H Gurung	'Nalwa Medal' by the Indian Red Cross Society. New Delhi.
5	NR-12184	Maj (Miss) PY Wad	'National Award for Nursing Personnel' by the Govt. of India, Ministry of Health and Family Planning (Deptt of Health) New Delhi.

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4	NR-20713	Brig (Miss) LI Braganza	23 May 73
5	NR-12116	Col (Miss) DBM Bhagwandas	29 May 73
6	N-680947	Col (Mrs) S Chacko	12 Jun 73
7	NR-12035	Lt Col (Miss) F J Sahayar	21 Nov 73
8	NR-12067	Lt Col (Miss) C Hutton	09 Dec 73

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4	MR-2803	Flt Lt ML Arora	15 Nov 73
5	DR-10061 L	Lt Col AK Sen	1 Dec 73
6	MR-195	Maj Gen SN Mukerji	12 Dec 73
7	DR-10063 W	Lt Col AB Marwaha	Dec 73
8	NR-14753	Lt (Miss) AV Rao	9 Mar 73
9	NR-15089	Lt (Miss) Pushpa Sood	16 Mar 73
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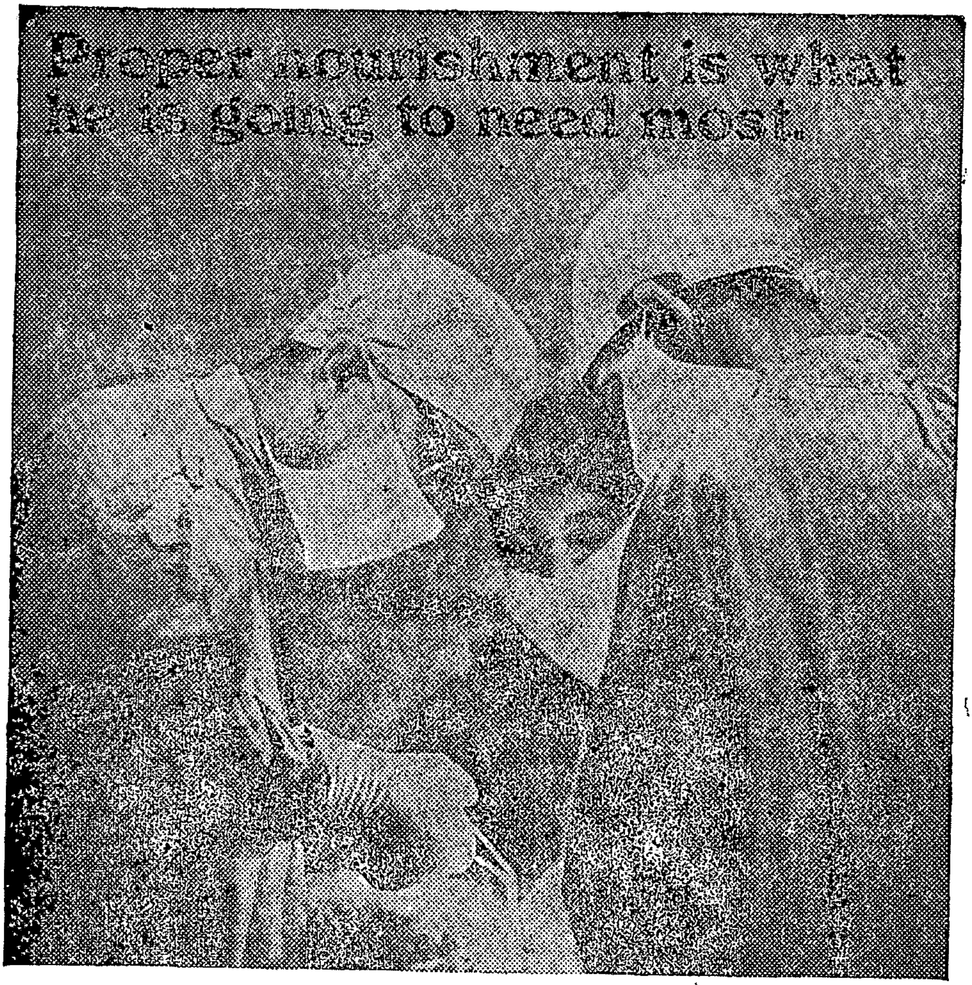
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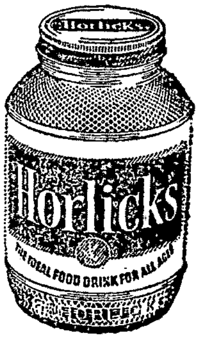

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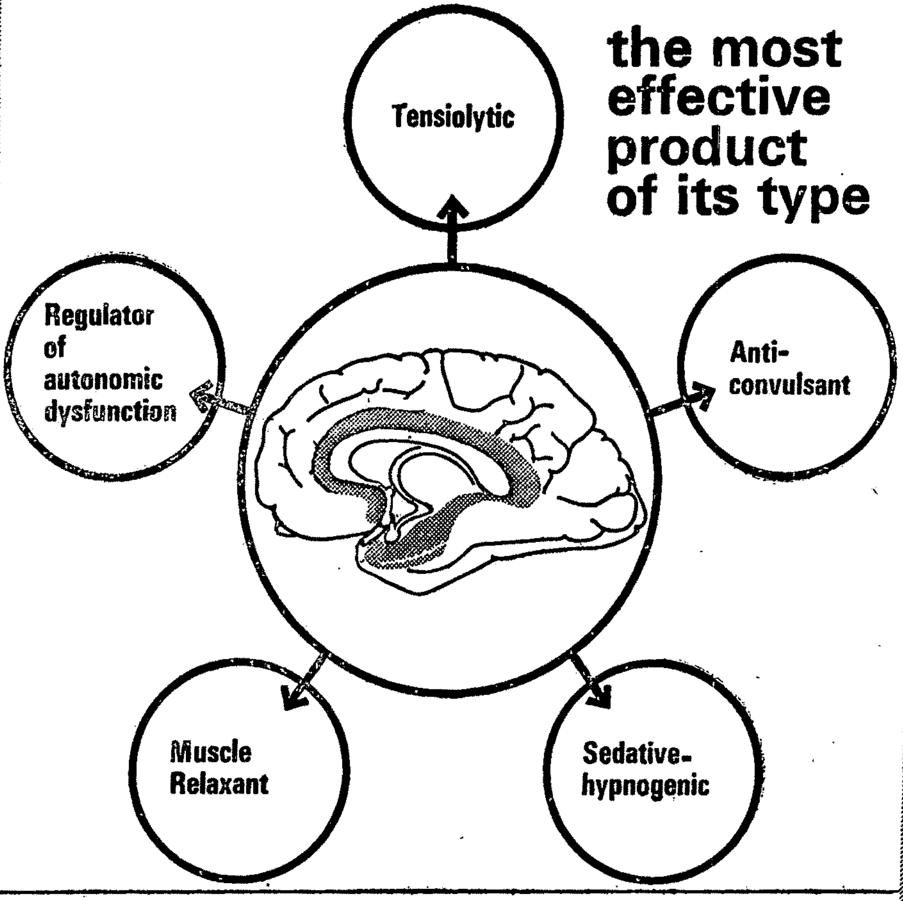

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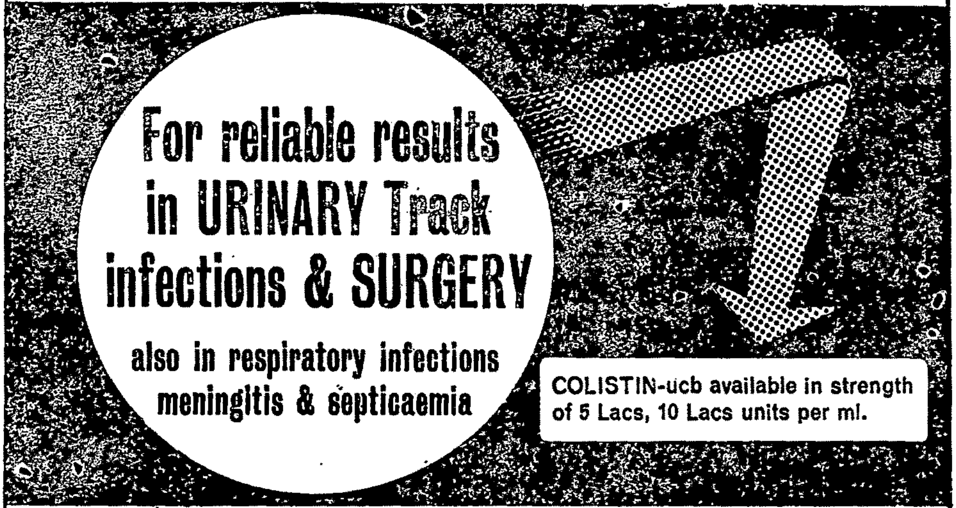

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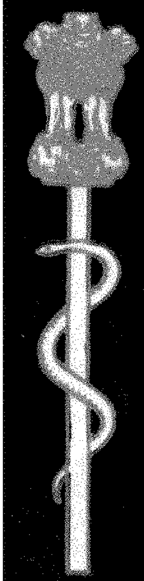

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EVALUATION OF HEALTH PROGRAMMES

BY

M. I. D. Sharma*

Evaluation has been defined as the "Process of determining the value or amount of success in achieving a predetermined objective".[1] Evaluation of any health programme, therefore, means the assessment of the degree of success which has been achieved at a particular point of time in relation to the long term objective set for the programme. It is just like the physician continuously evaluating the progress of a patient under his care which enables him to make changes in the course of treatment when considered necessary.

There is a tendency to have complete faith in the organisational and operational aspects of a programme and entertain hope that the set objective would be achieved during the period stipulated. Such an attitude is not scientific because proper assessment of the success achieved is essential to discover the weaknesses of the organization and shortcomings of the operational aspects of the programme to enable the executing agency to improve and incorporate remedial measures in time. At the time of planning of a particular health programme, it is very often seen that necessary provision for effectively undertaking evaluation activities is not made. Similarly little scope for flexibility is provided in running the programme with the result that it becomes difficult to introduce any changes if indicated by evaluation activities later. In the initial stages, therefore, the planners should see that there is specific provision for evaluation of the health programme and that its goals are so set that there is room for making changes or if necessary, the goals may be changed or period of their attainment may be altered accordingly.[2]

The evaluation process is in the form of an ascending spiral beginning with evaluation at each stage.[1] (Fig. 1).

Initially base line information on the proposed programme is obtained, then the goal of the programme is set, yardsticks for measuring the goal are determined, goal measured, the goal attaining activities are identified and put into operation and the

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effect of the goal on the total problem is appraised. The process is repeated in an ascending order till finally the ultimate objective is achieved. The procedure is similar to the one followed by the physician who diagnoses the disease of his patient, determines if the disease can be cured or only symptoms are to be relieved, finds out the manner by which the progress of the case is to be assessed, measures his progress, identifies the particular treatment which suits the patient, puts him under this treatment and notes the effect till finally the patient no longer requires attention from him.

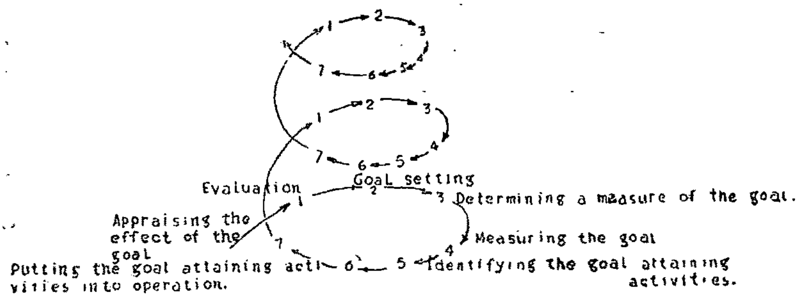
1
2
3
4
5
6
7
Evaluation
1
Goal setting
2
3 Determining a measure of the goal.
Appraising the effect of the goal
Measuring the goal
4
7
Putting the goal attaining activities into operation.
6
5 Identifying the goal attaining activities.

Fig. 1

PURPOSE OF EVALUATION

The evaluation of any health programme is usually carried out with the following objectives in view :

i. In planning for future, it is necessary to obtain base line information through evaluation on the achievements of the programme so that the future course of action is planned on a known base line.

ii. The other purpose of carrying out evaluation is regulatory in nature, ensuring the full and effective utilization of the staff and facilities of the programme.

iii. Similarly by identifying the weaknesses and the strong points in the technical and administrative aspects of a programme it should be possible for the evaluating agency to make such recommendations, so that the best use of the strong points is made and formulate and suggest corrective measures for the weak points to ensure that the programme is run on sound basis keeping the practical considerations in view.

iv. Evaluation helps also in examining the strategy of the programme so that necessary changes can be made in the administrative and operational aspects of the programme and if required the goals already set may be revised.

v. To get the full support of the concerned governmental agency for any health programme and to win the public opinion whose participation in the programme activities is essential, the achievements have to be determined, highlighted and made known to them.

ORGANISATIONS FOR EVALUATION

Executing Agency :

A properly run programme has some sort of machinery for its continuous evaluation so that the shortcomings are detected immediately on their occurrence and corrective measures to rectify the same are introduced as early as possible. Evaluation of programme by the executing agency is done through reports and returns which are received by it from its peripheral echelons. When prescribing the reports and returns to be submitted by the various levels of its operational wing, it should be seen that there is no duplication of information received and the submission of reports is kept to the minimum and only such items be included which are needed for evaluation. The other method by which the executing agency evaluates the programme is through the observations of its supervisory staff. For this to be effective it should be ensured that wherever required prompt action is taken by the executing agency on the observations of the supervisory officers.

Evaluation by outside agencies :

In addition to the programme organization itself the activities may be evaluated with advantage by an outside agency which can have an objective view of the whole thing. Such outside evaluation has been resorted to by the Malaria Eradication Programmes in different countries through independent appraisal teams comprising of national and international experts.

In some countries there may be an organization independent of the executing agency which can undertake such an evaluation of a programme as an outside agency. For example in India there is the Programme Evaluation Organitation in the Planning Commission which undertook the evaluation of the Family Planning Programme in the country in 1963-64 and again in 1968–69.

To ensure that all the aspects of the programme are evaluated properly, it is necessary to have a multi-disciplinary team consisting of epidemiologist, health administrator, biostatistician, social scientist and economist for the purpose.

PERIODICITY OF EVALUATION

i. Concurrent evaluation or progress reporting : In any health programme concurrent evaluation or progress reporting should be incorporated as its integral part so that evaluation of the programme is carried out simultaneously as the programme progresses. Concurrent evaluation is a routine assessment during the execution of the programme by the members of the organization as a part of the programme, achieved through progress reports. This is necessary so that important events are detected at the time of their occurrence instead of their being studied in retrospect. It provides information about the weaknesses of the programme which can be rectified immediately. It also discovers the strong points of the programme which may be further exploited to the fullest extent in the interest of the success of the programme. This concurrent progress reporting is a useful continuous feed back on the programme. This should cover all the aspects of the programme including consumers' response.

ii. **Periodical Evaluation :** Periodical evaluation is the evaluation carried out a the end of certain specified period, say, once a year, or two years etc.

iii. **Ad-hoc Evaluation :** Evaluation may be carried out at any point of time when it is so required on ad-hoc basis.

iv. **Terminal Evaluation :** Terminal evaluation is the evaluation undertaken when the programme is stated to have achieved its ultimate objective.

Provision for concurrent, periodical and terminal evaluations should be an essential component of overall planning. Periodical evaluation is carried out by agencies not directly connected with the execution of the programme who may view the performance in an objective and unbiased manner and may formulate suitable recommendations to improve the same. Similarly, evaluation may also be carried out as a terminal activity of the programme to find out whether the specified final objective of the programme has been reached when it is claimed by the programme implementing agency that it is so, say, the eradication of malaria or smallpox from a country or state. This too should be carried out by an outside agency (Fig. 2).

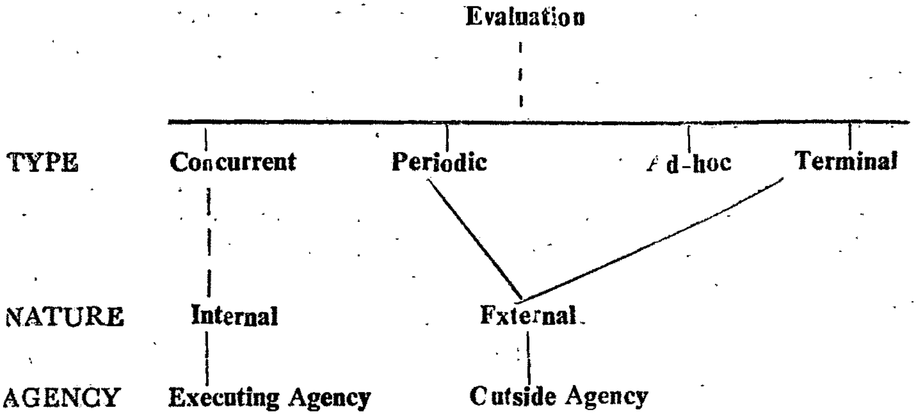

Fig. 2. Type, nature, periodicity of evaluation and agency for the same.

METHODOLOGY OF EVALUATION

In any health programme the ultimate objective has to be precisely defined. Similarly the initial and intermediate objectives are also to be stated clearly. For example in the Malaria Eradication Programme the ultimate objective is the complete elimination of morbidity and mortality due to this disease. The initial or starting objective should be to provide insecticide protective coverage to the population through spraying of houses which is the attack phase of the programme. Due to the interruption of transmission through insecticidal spraying of houses the parasitaemia in the community is reduced to such a low level that spraying of houses can be withdrawn and residual malaria cases can be picked up through surveillance operations. This is the intermediate objective or consolidation phase of the programme. When there is no indigenous transmission of the disease the ultimate objective is reached which is the maintenance phase of the programme when vigilance machinery ensures detection of any imported case or secondary introduced cases arising thereof.

The initial, intermediate and ultimate objectives are both performance and impact objectives at each level. For example, in the Malaria Eradication Programme the initial performance objective is spraying of all houses with residual insecticides and collection of blood smears from all fever cases through passive and active surveillance operations. The result of these operations would be interruption of transmission of malaria which is the initial impact objective. During the consolidation phase the intermediate performance objective is collection of bloodslides of all fever cases for detection and radical treatment of residual malaria cases, and the impact objective would be the absence of disease in the protected population. When the ultimate objective is attained that is during the maintenance phase the indigenous population would be free from malaria and this freedom from the disease would be kept up through strict vigilance on the immigrants, detecting and radically treating any malaria cases found in them or the introduced cases and providing insecticide protective coverage to such groups of population.

From operational point of view the objectives at each level are broken down to sub-objectives in terms of time, space and different categories of personnel. (Fig. 3.)

Attack phase 3-4 years	↓	**Initial performance objective :** Insecticidal spraying of all houses. Detection and radical treatment of malaria cases through collection of blood slides of all fever cases. **Initial impact objective:** Interruption of transmission in the community.
Consolidation phase 2-3 years	↓	**Intermediate performance objective:** Detection and radical treatment of all residual malaria cases. **Intermediate impact objective:** Absence of disease in the community.
Maintenance phase	↓	**Ultimate performance objective:** Vigilance on immigrants and detection and radical treatment of malaria cases in them. **Ultimate impact objective:** Disease - free status of the community assured.

Fig. 3. Showing the objectives in the Malaria Eradication Programme.

STEPS IN THE EVALUATION OF HEALTH PROGRAMME

i. **Collection of base-line data with regard to the specific programme in the community :** This is essential for the purpose of comparing the situation after a given period of time which in turn indicates the extent of success achieved through the implementation of the specific health porgramme. The data required for this purpose will be in terms of morbidity, mortality due to the disease, age and sex specific rates; demographic data etc. with special reference to the programme under implementation. In the case of the Malaria Eradication Programme, it would be the incidence of disease in the population to be brought under the programme.

ii. **Statement of goals to be achieved in the particular health programme :** A clear and precise statement regarding the objectives, classified as initial, intermediate and ultimate objectives and further broken down to sub-objectives leading to individual activities for the execution of the programme should be made. This is done before the start of the actual operations of the programme during the planning stage.

iii. **Determining the indices to measure success :** This is another important aspect in the process of evaluation. After having collected data about the programme if these are to be made use of in a proper way so as to measure the change the activities have brought about in the disease status, the indices serve as invaluable tools. For example in the Malaria Eradication Programme, the ultimate objective is the complete elimination of morbidtiy and mortality due to malaria in the community. The indices that need to be worked out are to measure morbidity and mortality due to malaria. This can-be achieved through incidence and death rates. To attain the ultimate objective the measures taken include the provision of insecticide protrective coverage to the community and detection and radical treatment of residual malaria cases through surveillance operations. The measurments which may be used in this case are the percentage coverage of houses sprayed and the annual blood examination rate of community. These give information on insecticidal spray operations and surveillance operations respectively.

iv. **Measurement of success in terms of the value of data collected:** Having obtained the data by using the relevant indices for a particular programme, the next step is to measure the degree of success achieved. This is particularly imprortant in the case of a disease eradication programme where the progress from one phase to the next one is dependent on these data. For example, in the Malaria Eradication Programme an area would enter from attack into the consolidation phase if the incidence of disease is less than 100 cases per million population. Similarly if there are no indigenous cases of malaria in the population for two to three years the area would enter from the consolidation into the maintenance phase.

v. **Study design for appraising the degree of success in the implementation of the programme:** From the data obtained it is necessary to elicit the information about the degree of success in a particular programme and for this purpose it is essential to have a proper study design. Taking the example of disease eradication or control programme there are two main types in which the study may be designed.

In one case similar data are obtained from the programme area and also from a comparable index area and thus measure the success due to measures implemented in the former area. Such a design has the handicap that it is not always easy to have an index area which is comparable in all respects with the programme area and secondly it is not possible to exercise such a control that no measures or inputs are introduced in the index area so that it may continue to serve as a proper comparison area. Such a practice is some time adopted in pilot studies for determining the effectiveness of particular disease control/eradication measures.

In the other design comparison is made between similar data collected before and after the implementation of a particular programme. For example, in the case of a disease eradication or control programme, the incidence or prevalence rates of the disease may by compared before and after the implementation of the programme and from this the degree of success achieved may be assessed. This design too has a disadvantage that due to improved diagnostic facilities with the passage of time and with efficient case detection machinery more cases may be detected than earlier. Therefore, the particular design which may be used in respect of a health programme would depend on the nature of the programme keeping in view the limitations stated above.

vi. **Evaluation of the administrative aspects of the programme:** The administration of a programme has a direct bearing on its success. Therefore, in the evaluation of a health programme, it is necessary to have information on the various aspects of its administration.

(a) **Organisational aspects:** For evaluating the suitability and adequacy of the organisation information on the following should be collected :

1. Staff of different categories required, sanctioned and in position :
2. Staff turnover, category-wise :
3. Job description of individual workers :

4. Training of staff :
5. Work expected to be performed and actually performed :
6. Morale of the staff.

(b) **Managerial aspects :** The human relation aspects of the organization may be assessed by having information on communication, supervision and team work. It may be seen that the channel of communication is short and supervision is of the supportive type preparing the supervisees for team-work.

(c) **Logistics aspects :** Information may be obtained to see whether or not the supply of equipment, material and transport is adequate and timely particularly in cases where these are to be imported. It may also be ascertained if there are proper and adequate arrangements for the maintenance and repair of equipment and transport.

(d) **Fiscal aspects :** It may be seen that the funds provided for the programme are adequate and that there is sufficient decentralisation of financial powers at different levels so that the operational efficiency of the organisation is promoted.

vii. **Discussion on findings and recommendations for action :** The evaluation activity in respect of a programme would only be complete after the observations have been discussed, properly analysed and interpreted which would result in the formulation of suitable recommendations for action. In making the recommendations following points should be kept in view.

(a) What has been the degree of success as indicated by the data, collected in relation to the stated objectives of the programme ?

(b) Has the programme progressed in accordance with the laid down time schedule ? If not what particular problems hampered the progress and what action is needed to tackle them ?

(c) Make a list of the strong points of the programme and indicate as to how best they can be exploited in the interest of the programme.

(d) Similarly make a list of the weak points discovered in the programme with suggestions to remedy them.

(e) Make a list of the technical and operational problems which need solution for the efficient implementation of the programme. Suggest some research studies and field investigations which may help in decision-making to solve the problems.

(f) Make a list of the problems needing attention in the administrative aspects of the programme with suggestions for removal of such administrative bottlenecks.

(g) For the proper implementation of the programme, is there need to change policies and procedures ? If so indicate such policies and procedures which require change with the precise nature of change for each.

DIFFICULTIES IN THE EVALUATION OF HEALTH PROGRAMMES

Following are some of the difficulties which may be encountered in the evaluation of health programmes :

i. If in the initial planning stage specific provision is not made for efficien concurrent evaluation, it becomes difficult to assess the achievements of the programme as it progresses. Similarly if there is no provision for the periodical evaluation of the programme it is not possible to know the degree of its success at a particular point of time. In the same manner in the planning stage itself provision for terminal evaluation should be made so that the claim of the executing agency of having achieved the ultimate objective is substantiated by an outside agency. It follows, therefore, that those matters should be taken care of in the planning stage itself.

ii. The executing agency as well as the administration responsible for a particular health programme should have an "Evaluative Mentality". Those responsible for the execution of the programme should consider evaluation activities "Non–Threatening" and of help to them in improving the operations. Similarly those responsible for the administration of the programme should not only see the shortcomings detected but should also note the achievements of the programme and give due weightage to both particularly giving necessary credit to the executing agency for what has been achieved.

iii. In the health field, simple tools and know-how are not available for assessing the economic benefits accruing from a particular health programme. There is urgent need to develop these so that the administrators are convinced that there is more than fair return from the investment on the programme.

CONCLUSION

In conclusion it may be stated that the evaluation of a programme provides base-line information at a particular point of time when it is carried out, it gives information on the past activities and indicates the course of action which may be followed in future with advantage.

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STUDY OF RADIO-ERYTHRO-FERROKINETICS IN THE MANAGEMENT OF NORMOCYTIC HYPOCHROMIC ANAEMIA (INCLUDING HAEMOLYTIC ANAEMIA) -AN EVALUATION OF DIAGNOSTIC, THERAPEUTIC AND INVESTIGATIVE SIGNIFICANCE

AFMRC PROJECT NO. 337/68

BY

Lt Col R.N. Banerjee*

ABSTRACT

An evaluation of the diagnostic, therapeutic and investigative (research) significance of radio-erythro-ferrokinetics has been carried out in a study of 477 patients presenting normocytic hypochromic anaemia, refractory to routine haematinic therapy. The study has revealed four major aetiological groups of such anaemia viz. (i) congenital haemolytic disorders, (ii) acquired immuno-haematological disorders, (iii) secondary anaemia of malignant and non-malignant systemic diseases and (iv) chronic nutritional disorders. The study, one of the most extensive survey published so far and first of its kind in our country, has convincingly established the values of the radio-erythro-ferrokinetics in the quantitative assessment of red cell kinetics in haemolytic and dyshaemopoetic disorders of primary and secondary aetiologies. The nuclear methodologies have been found useful in arriving at definitive diagnosis, in assessing therapeutic approaches and in understanding the aetiopathogenesis of anaemias of obscure origin. The results of the study suggest that the two nuclear methodologies deserve to be introduced in the large referral hospitals in our country for scientific as well as economic reasons in the management of anaemia, which is a common clinical disorder in our population.

INTRODUCTION

Normocytic anaemia often presents clinical problems in its diagnosis and treatment. The problem becomes formidable, when such anaemia proves refractory to the routine haematinic therapy. Lack of precise diagnostic clues leads to prolonged morbidity and hospitalisation. In such cases, therefore, it becomes necessary to obtain more detailed information about the possible aetiopathogenesis of the anaemia, since such anaemia state may be the clinical presentation in variety of disorders with primary or secondary dyshaemopoesis or haemolysis. In investigating the aetiopathogenesis of such dyshaemopoetic state, a quantitative evaluation of the red cell kinetic with respect to its rates of erythropoesis, maturation, bone-marrow release and rate and sites of destruction, promise to be of valuable aid in an early diagnosis and treatment of the cases.

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During the last two decades, the introduction of radio-tracer techniques in the erythro-ferrokinetic problems has facilitated such investigations. Anaemia either of nutritional or other aetiology is one of the commonest causes of morbidity among our population. The present work was undertaken to evaluate the significance of radio-erythro-ferrokinetic in normocytic anaemia of common occurance in our country. As far as the author is aware this is one of the most extensive evaluating study of radio-erythro-ferrokinetic in the relevant literature and a first report of its kind in this country.

MATERIALS AND METHODS

(i) **Clinical Material** : Four hundred seventy seven patients of anaemia, attending the Haematology and Nuclear Medicine clinics of Safdarjang Hospital during the period of 1968-72 were studied. The diagnosis in each case was established by clinical, laboratory, radiological and radio-isotopic investigations. The major criterion of selection of cases has been the predominant normocytic anaemia, resistant to routine haematinic therapy.

(ii) **Haematological studies** : The routine haematological studies such as complete haemogram, platelet count, reticulocyte count, erythrocyte sedimentation rate, red cell osmotic fragility test, direct antiglobulin test, bone-marrow examination by sternal puncture and relevant cyto-chemical studies (Sudan Black, Peroxidase, PAS and Foulgen staining) were done by standard methods as described by Lacie and Lewis (1968).

(iii) **Biochemical Studies** : Serum iron was estimated by the method of Ramsay (1957). Supportive biochemical investigations for renal and liver function tests were done by standard colorimetric methods.

(iv) **Radiological Investigations** : Supportive radiological investigation such as skeletal survey, intravenous pyelography, Barium meal examination, splenoportal venography and chest X-rays were carried out in relevant cases. In selected cases of lymphoproliferative disorders abdominal lymphangiography was carried out.

(v) **Biopsy Examination** : Lymphnode and tissue biopsy examination was done in all cases of Reticulosis and non haematologic malignancy. The final diagnosis of portal cirrhosis was made by liver biopsy examination by the standard procedure.

(vi) Radio–isotopic studies :

(a) Radio–chromium erythrokinetic studies :– Radioactive Chromium (Na_2 $^{51}Cr\,O_4$) supplied by Bhaba Atomic Research Centre, Bombay, was used to label human red cells for the determination of autologous and homolgous red cell survival time (^{5} Cr RBC T$\frac{1}{2}$) in patients and index of splenic sequestrations of labelled red blood cells, according to the methods described by King and Mitchall (1961).

(b) Radio–chromium–ferrokinetic studies :– Radioactive iron ($^{59}Fecl_3$) received from Bhaba Atomic Research Centre, Bombay, was injected in the form of sterile ferric citrate complex, pH 6, intravenously to the patient, to determine the (i) plasma ^{59}Fe disappearance half time (PID–T$\frac{1}{2}$), plasma iron turnover rate (PITR), red cell ^{59}Fe utilization rate as described by Huff, Hennessy, Austin, Garcia, Roberts and Lawrence (1960). Organ localisation of ^{59}Fe and its disappearance or building–up rate was determined by in–vivo counting over the sacrum, liver, spleen and heart for 10 days after the injection of ^{59}Fe solution.

(c) Radio-cobalt (^{58}Co) labelled Vitamin B^{12}, supplied by Bhaba Atomic Research Centre, Bombay, was employed in measurment of intestinal B^{12} absorption rate by the method described by Schilling.

(d) Radio-hippuran Renography : Radio–iodinated (^{131}I) hippuran, received from BARC, Bombay was used in carrying out the radio hippuran renography in cases of acute and chronic renal failure according to the method described in our earlier publication (Banerjee et al, 1971).

(e) Radioactive rose–bengal excretory hepatogram :– Radio–iodinated (^{131}I) rose–bengal, obtained from BARC, Bombay, was employed in radio–isotopes excretory hepatogram in cases of portal cirrhosis or anaemias of suspected hepatic origin.

RESULTS

I. Aeatiology of normocytic anaemia :

Table-1 shows the aetiological distribution of 477 cases studied in the present series. For the purpose of the clinical and haematological correlationship, an arbitrary grading of the anaemia was adopted on the criterion of the haemoglobin values. The anaemia was considered to be mild, moderate and severe according to the haemoglobin values below 12,10 and 6 gm per 100 ml. respectively.

Table 1

Normocytic Hypochromic Anaemia Studied by Radio-Erythroferrokinetic during the Year 1968 to 1972

Aetiological Group		Diagnosis	No. of cases Studied
I. Congenital Haemolytic anaemia	(a)	Congenital spherocytosis	8
	(b)	Non-spherocytic Haemolytic Anaemia	6
	(c)	Thalassaemia Major & Minor	65
II. Acquired Haemolytic Anaemia		Auto-immune & Isoimmune Type	16
III. Secondary or Symptomatic Anaemia	(a)	Malignant Reticulosis	25
	(b)	Chro Myeloid & Lymphatic Lukaemia	20
	(c)	Myeloseclerosis with Myeloid Metaplasia	4
	(d)	Multiple Myeloma	4
	(e)	Non-haematologic Malignancy	42
	(f)	Systemic Lupus Erythematosis	1
	(g)	Portal Cirhosis	54
	(h)	Acute & Chronic renal failure	32
	(i)	Anaemia of Burns	52
	(j)	Hypersplenic Pancytopaenia	3
	(k)	Hypoplastic and Aplastic Anaemia of primary and secondary aetiology	27
	(l)	Paroxysmal Nocturnal Haemoglobinuria (PNH)	4
IV. Nutritional Anaemia	(a)	Iron Deficiency Anaemia	85
	(b)	Megaloblastic Anaemia	7
	(c)	Hypothyroid Anaemia	15
	(d)	Idiopathic steatorrhoea	6
	(e)	Pyridoxin Deficiency	1
		Total	477

II. Study of Diagnostic Significance :

(a) Radio Erythrokinetic in haemolytic disorders. Table-2 shows the results in 165 cases of anaemia of varied aetiology.

For the purpose of this study, the mean normal values of ^{51}Cr-RBCT$\frac{1}{2}$ (Days) and splenic sequestration index (Spleen : Liver ratio on T$\frac{1}{2}$ (day) were taken to be 28.5

Table 2

Diagnostic Significance of Radio – Erythrokinetic

(Total No. of Cases 200)

S. No	Diagnosis	Autologous Radio-erythrokinetic study			Homologous Radio-erythrokinetic study		
		No. of cases	Mean ^{51}Cr-RBCT$\frac{1}{2}$ (days)	Mean Sp. Liver Ratio onT$\frac{1}{2}$ (days)	No. of cases	Mean ^{51}Cr-RBCT$\frac{1}{2}$ (days)	Mean spleen Liver ratio on the (T$\frac{1}{2}$ (days)
1	Congential Spherocytic Anaemia.	8	18.5	3.0	3	26.5	1.2
2	G-6P. D. Def. and other Non-Spherocytic Haemolytic Anaemia.	6	14.0	2.8	4	27.8	1.4
3	Secondary Haemalytic Anaemia of varied aetiology						
	(a) Burn	35	20.0	2.0	15	18.5	2.0
	(b) Portal Cirrhosis	32	14.5	2.5	5	18.0	2.8
	(c) Renal Failure (acute & chronic).	22	16.0	1.8	6	16.5	1.8
	(d) Malignancy (non-haematological).	22	24.0	1.8	8	22.5	2.0
	(e) Lukaemia (both myeloid & Lymphatic).	14	18.5	2.2	5	18.0	2.4
	(f) Lymphoma.	25	7	3.1	8	12.0	3.0
	(g) Primary Hypersplenism.	1	14.0	4.5	1	14.5	3.5
	(h) Iron Def. Anaemia.	35	10.9	1.3	7	24.5	1.4

days (range 25.5–32 days) and 1.5 (range 0.9–1.8) respectively as reported in our earlier publication (Banerjee and Narang, 1967). The series included 14 cases of primary haemolytic state and 151 cases of overt or latent haemolytic state of secondary character. In 62 cases the radio-erythrokinetic study was repeated after a period of 4-8 weeks with homologous blood group matched Blood Bank normal red blood cells. The results are shown in table-2.

(b) Radio-ferrokinetic in dyshaemopoeitc anaemia. Table-3 shows the results in 163 cases of anaemia of dyshaemopoesis of varied aetiology. Eighty five cases of iron–deficiency anaemia of prolonged duration for period over three months, were included in the study. The cases presented a predominantly normocytic hypochromic anaemia with mild to moderate microcytosis.

Table 3

Diagnostic Significance of Radio-Ferrokinetic in Refractory Anaemia

	Diagnosis	No. of cases	PIDT$\frac{1}{2}$ (Minutes)	PITR(mg/100ml Plasma/day
1	Iron Def. Anaemia	85	32	0.38
2	Drug induced Aplastic Anaemia			
	(i) Chloromycetin	18	140	0.4
	(ii) Novalgin	2	135	0.42
	(iii) Butazolidin	1	145	0.38
	(iv) Neomercazol	2	140	0.40
3	Preleukaemic Aplastic Anaemia	5	165	0.35
4	Terminal Leukaemia	3	155	0.30
5	Non Haematological Malignancy	35	145	0.42
6	Siderocrestic	2	130	0.58
7	PNH-Aplastic Anaemia	4	140	0.42
8	Portal Cirrhosis (Iron deficiency anaemia) with occult G. I. Bleeding	22	90	0.8
9	MMN (Myelasclerosis Myeloid Metaplasia)	4	105	0.52

The mean normal values of PIDT$\frac{1}{2}$ (Minutes) and PITR (mg/100ml/day) of plasma were taken to be 95 minutes (range 80-120 minutes) and 1.245 mg/100ml plasma/day (range 0.35 – 3 mg/100 ml plasma/day respectively as reported in our earlier publication (Banerjee, and Narang, 1967).

III. Study of Therapeutic Significance :

(i) Radio–erythrokinetic in the evaluation of hypersplenism and selection of cases for therapeutic splenectomy.

Table 4

Therapeutic Significance of Radio Erythro-Ferrokinetic in the Management of Normocytic Anaemia :

I Erythrokinetic in Splenectomy

(No of Cases 26)

Diagnosis	No of cases investigated	Preoperative ^{51}Cr–RBC–T$\frac{1}{2}$ (days)	findings Sp. Seq. Sp. Liver	(Sp. Liver) Post Opperative findings ^{51}Cr RBC–T$\frac{1}{2}$ (days) (No of cases in paranthesis)
1. Congenital Sphrocytic.	6	10.5	3.5	24.5 (3)
2. Non. Sph. Haemolytic Anaemia.	6	16.0	3.2	26.0 (2)
3. Thalassemia.	4	10.0	3.0	21.0 (2)
4. Lymphoma.	2	17.5	2.8	22.0 (1)
5. Primary Sp. Neutropaenia.	1	11.5	3.0	26.0 (1)
6. Portal Cirrhosis	7	16.5	2.8	24.0 (5)

Table 5

Therapeutic Significance of Radio Erythro–Ferrokinetic : II Erythrokinetic in Steroid Therapy
(No of Cases 65)

Diagnosis	Before Therapy		Mean Hb gm/ 100 ml.	After Sex weeks of steroid Therapy		
	No. of Cases	Mean ^{51}Cr–RBC–T$\frac{1}{2}$		No of cases	Mean ^{51}Cr–RBC–T$\frac{1}{2}$	Mean Hb gm/100ml.
1. Autoimmune Haemolytic Anaemia of varied aetiology.	16	14.5	6.5	12	22.0	10.0
2. Haemoglobinopathy (Thalassaemi)	35	12.5	5.0	28	18.0	7.5
3. Hypoplastic Anaemia	12	20.5	7.5	8	24.0	9.5
4. PNH–Aplastic Anaemia.	2	11.0	5.5	2	18.0	7.0

Table 6

Radio-Ferrokinetic in Normocytic Hypochromic Anaemia of Nutrient Deficiency
(No of Cases 77)

Diagnosis	No of Cases	Therapy	Ferrokinetics & Hb. Conc.							
			Before Therapy				After Therapy (After 6 weeks)			
			Serum Iron mg per 100 ml	PIDT½ minutes	PITR mg/100ml plasma	Hb gm%	Serum Iron mg per 100 ml	PIDT½ minute	PITR	Hb gm%
1. Nutritional Iron Deficiency.	40	Intravenous Iron (Imferon)	45	28	3.4	4.5	92	82	1.8	9.5
2. Portal Cirrhosis with occult melena.	15	-do-	54	60	1.2	7.5	110	130	0.8	10.0
3. Malabsorption Syndrome.	6	-do-	60	32	2.4	6.0	80	92	1.6	9.5
4. Pyridoxine Def. Anaemia (Siderocrestic)	1	Oral Pyridoxine	130	145	0.8	5.5	115	110	1.6	9.0
5. Hypothyroidism	15	Oral Eltaroxin	140	135	0.4	8.5	110	90	2.4	11.2

Table 7

Results of Radio Erythroferrokinetic in Secondary Anaemia
(No of cases 323)

Clinical State	No of cases	^{51}Cr–Erythrokinetic value. (Mean)			^{59}Fe Ferrokinetic value (Mean)		Inferance (Type of Anaemia)
		No of cases	RBC T½ (days)	Spleen Liver	No of cases	PIDT½ Min	
1. Anaemia of Burns Acute (Ist Week)	52	22	18.0	—	18	40	Haemolytic Post Haemorrhage.
Chronic (After 12 Weeks)	—	13	20.5	1.8	10	135	Hypoplastic anaemia with mild hypersplenism.
2. Renal failure	32	22	16.0	1.6	10	142	Haemolytic state with bone–marrow depression.
3. Portal Cirrhosis	54	32	14.5	2.4	22	84	Predominant Haemolytic hyper splenisim.
4. Lukaemia (Myeloid Lymphoid)	20	20	22.5	1.8	—	—	Suggestive of reduced red cell life span, latent haemolysis.
5. Lymphoma.	27	27	20.5	2.2	4	135	Latent Haemolytic Anaemia.
6. Non–Haematological Maliganancy.	42	26	22.0	2.2	20	65	Mild haemolytic with extra medullary iron shunt.
7. Thallassaemia.	65	50	12.5 (8 to 19)	2.0 (1.6 to 3.5)	15	32 (20 to 65)	Haemolytic selective hypersplenism & extra medullary erythroposesis.
8. Anaemia of bonemarrow depression including primary or secondary aplastic anaemia.	27	24	20.5 (12 to 28)	1.8 (1.2 to 2.0)	22	145 (122 to 180)	Poor Erthropoesis MILD Haemolytic state.
9. Paroxysmal Nocturnal Haemoglobinouria.	4	3	10.5 (9 to 14)	1.5	2	138 (125 to 155)	Haemolytic with marrow dyshaemapoesis.

Organ localisation shows significant extramedullary erthropoesis in spleen in all severe cases.

(ii) Radio–erythrokinetics in the evaluation of steroid therapy in haemolytic anaemia and anaemia of dyshaemopoesis.

Table-4 shows the mean results of pre–operative assessment in 26 cases of primary and secondary hypersplenism and the post–operative follow–up results in 14 cases. The remaining 12 cases were not available for follow–up study.

Table–5 shows the mean results of ^{5} Cr–RBCT$\frac{1}{2}$, with their corresponding haemoglobin values, in 65 cases of anaemia. Out of 65 cases evaluated before starting the therapy, 50 cases were available for complete follow–up studies.

(iii) Radio–ferrokinetic in the evaluation of specific haematinic therapy in normocytic hypochronic anaemia of nutritional deficiency.

Table–6 shows the results in 77 cases of the mean values, before and after treatment in anaemias of iron deficiency, portal cirrhosis with occult melena, malabsorption syndrome,pyridoxine deficiency and hypothyroid state.

IV. Combined Radio–erythro–ferrokinetic study :

Table–7 shows the mean values of radio–erythro–ferrokinetics in 323 cases of anaemia of varied aetiology. The duration of the combined investigative study has been of three to four weeks.

DISCUSSION

Anaemia of varying degree is a common presentation in many clinical disorders. While in nutritional anaemia, the micro or macrocytic morphological alterations in the erythrocytic size, give an aetiological indication, this diagnostic feature is absent in normocytic anaemias. The normal sizes and the minimal alterations in the haemoglobin content of the erythrocytes in such disorders generally signifies a reduction in the circulating red cell mass either due to increased destruction or impaired rate of production, independant of the supply of erythropoetic materials. The rational therapeutic approach to such problems consequently requires an assessment of the state of erythrokinesis and bone–marrow haemopoesis. Since the introduction of the radioisotopic tracer techniques, it has been possible to apply these methods in the mangement of such haematological disorders. The present study was undertaken to evaluate the diagnostic, prognostic and investigative significance of these methodologies in cases of normocytic anaemias commonly observed in our patient population.

Aetiology of normocytic anaemia :

In the present series the aetiology of the normocytic anaemia could be broadly divided into four groups viz. (i) the congenital haemolytic disorders, (ii) the acquired immuno–haematological disorders, (iii) the secondary anaemia accompanying systemic diseases of malignant and non–malignant nature and (iv) the chronic nutritional disorders. Because of the pre–selective critieria, the incidence for the disorders will have no epidemiological significance.

The congenital haemolytic anaemias usually present the classic presention of refractory chronic anaemia of mild degree with periodic exacerbations due to haemolytic crisis. Often such cases have been confused with episodic incidence of viral hepatitis because of the accompanying icterus. However, the diagnosis can often be established by standard haematological examinations. The difficulties in diagnosis mainly arise in cases of non-spherocytic haemolytic anaemias.

The anaemias of immuno-haematological disorders often present diagnostic difficulties particularly if the direct or indirect antiglobulin tests do not yield conclusive results. The anaemia may appear de-novo as primary disorders or secondary to iso-antibodies resulting from repeated blood transfusions.

The third aetiological group forms a major study in the present work. In recent years, attention has been drawn to the haemolytic and dyshaemopoetic nature of secondary anaemia accompanying varieties of chronic disorders. It is becoming increasingly clear that in the management of such disorders, besides the specific treatment for the primary aetiology, necessary care has also to be taken of the haematological problem accompanying the disease. The examples of such incumbancies are brought out clearly in the management of malignancy, systemic lupus erythematosis, portal cirrhosis, acute or chronic renal disease, burns etc. In the present study, this group has shown particularly the usefulness of radio-erythroferrokinetics in discerning the exact mechanism of the anaemia in the disorders.

It is a common thinking that the nutritional anaemias would present a clear cut microcytic or macrocytic anaemia arising from iron or B_{12}-folic acid deficiency. A striking number of cases, in the present series were diagnosed to be of nutritional deficiency origin, who predominantly presented a normocytic hypochromic anaemia. The exact nature of the anaemia could only be established by detailed haematological and radioactive Vitamin B_{12} absorption studies. All the cases were of anaemia of long duration and the causes of their normocytic presentation could be attributed to either inadequate haematinic therapy or supplementary haematinic therapy without treatment of the primary aetiology. This was particularly noticed in cases of chronic iron deficiency anaemia of chronic bleeding disorders in men and women, ankylostomiasis in children and gastro intestinal malignancy or portal cirrhosis in elder age groups.

Cr^{51} Radio-Erythrokinetic :

A physiological incorporation of radioactive chromium (^{51}Cr) in the globin molecule of the erythrocyte provides a suitable method for labelling the erythrocytes for the study of erythrokinesis. In any such study, the essential information that one can derive is of the red cell life span, its rate of disappearance from the circulation and the major sites of its destruction.

In Primary Haemolytic Anemia :

In the present study, the results of Cr^{51} Erythrokinetic study in well established cases of haemolytic anaemia of known aetiology such as in congenital spherocytic anaemia and haemolytic anaemia due to G-6-PD deficiency or haemoglobinopathy, showed a significant reduction in the life span of patients own red cells (autologous). This has confirmed the diagnostic value of such study.

In such cases, with mild to moderate degrees of anaemia, a mean RBC T½ of 14 to 18.5 days have confirmed that the reduction in the red cell mass is essentially due to a rapid rate of red cell destruction. The concomitant study of splenic sequestration, showing the higher rate of radioactivity trapping in the spleen confirms the role of spleen as the predominant site of destruction of the erythrocyte. However, it is clear that in such cases the cause of the increased rate of erythrocyte destruction is not splenic but inherent deficiencies in the erythrocyte itself. This fact is borne out by the results that in such cases, while the ^{51}Cr RBC T ½ is reduced with autologous cells, the life span remains normal with transfused homologous normal red cells. (Table 2).

The significance of these observations have been examined by repeating the studies in the patients after therapeutic splenectomy. (Table 4). Since it is known that in the present state of our knowledge, we have no method of correcting the inherited deficiencies in the erythrocytes as in congenital spherocytosis, haemoglobinopathy or in enzyme deficiency anaemia, it is expected that the patients may derive benefit of prolonging the erythrocyte life span by removing the predominat site of its destruction. In 16 cases of haemolytic anaemia (Table-4), a normal or near normal prolongation of erythrocyte life span was restored by splenectomy. In our studies, in selecting cases for therapeutic splenectomy, two major criteria were found important. A ^{15}Cr RBC-T½ of less than 20 days and a splenic sequestration rate as measured by the spleen: liver ratio on the T½ day, of above 2.8 was found to be the most satisfactory criteria. The advantage of therapeutic splenectomy was however observed to be limited in cases of haemoglobinopathy or auto-immune haemolytic anaemia, where the results were less satisfactory, than in cases of congenital spherocytosis.

Of the less common causes of haemolytic anaemia we have examined the results in one case of primary splenic neutropaenia and two cases of Paroxyomal Nocturnal haemoglobinurea- Aplastic anaemia syndrome. It is known that in such disorders, the haemolytic anaemia is accompained by a significant bone-marrow dyshaemopoesis. Therefore the information derived from ^{51}Cr erythrokinetic studies in such cases would only be partial. In both the cases in the present series, the results of erythrokinetic studies satisfied the criteria of therapeutic splenectomy. The follow-up study showed that while in the case of the Primary splenic neutropaenia, there was a near normal (26 days) restoration of RBC-T½, in cases of PNH-Aplastic anaemia syndrme, there was only a marginal (^{51}Cr RBC T1/2 from 11 to 18 days) benefit and the morbidity continued primarily due to bone-marrow dyshaemopoesis.

Secondary Haemolytic Anaemia

In recent years, the state of latent haemolytic anaemia has been recognised in a variety of clinical disorders. It has been observed that in such conditions the usual diagnostic features of haemolysis such as reticulocytosis, bilirubinaemia, or gross anisopoikilocytosis are more often absent. The haemolytic nature of the accompanying anaemia, refractory to treatment with haematinics, can however be established by ^{5}Cr Erythrokinetic study. In the present series, the detection of the secondary haemolytic anaemia was specially investigated in 185 case of burns, portal cirrhosis, acute and chronic renal failure, non haematological malignancy, myeloid and lymphatic lukaemia, lymphoma and in chronic iron deficiency anaemia.

Gross reduction in the red cell life span was observed in lymphoma, portal cirrhosis, renal failure and lymphatic lukaemia. Significant, though mild to moderate reductions were observed in cases of burns, non-haematological malignancy and iron deficiency anaemia. Further investigations revealed that only in about 30% of the lymphatic lukaemia, the reduction could be attributed to autoimmune antibodies, while in the other cases the cause could be either corpuscular deficiency or mild to moderate hypersplenism. The repeated radio-erythrokinetic studies with homologous labelled red cells, have proved a definitive differential diagnostic aid in such problems. As shown in table-2, while the reduced red cell life span with labelled autologous red cells can be corected by the normal span of transfused labelled homologus red cells in anaemias of corpuscular defect, the shortening of life span remains unaltered with the homologous cells in cases of extra corpuscular factors. In such cases a further differentiation between the haemolytic state due to the plasma factor and the splenic factor can be arrived at from the high splenic sequestration indices in the latter group of disorders. Such hypersplenism was conspicuous in cases of portal cirrhosis, malignant lymphoma and primary splenic neutropaenia where a therapeutic splenectomy may be of benefit.

Studies of reduced red cell life-span in chronic iron deficiency anaemia is an interesting and significant observation. Results of normal life span of transfused homologous red cell in the subjects of iron deficiency anaemia provided a conclusive evidence that in such cases, the haemolysis is due to corpuscular deficiency, the exact nature of which remains obscure, although it could be attributed to iron deficiency itself.

Results in portal cirrhosis shows that in well over half of the cases, there is a significant hypersplenism and the results of splenectomy followed by porta-caval shunt have been found to be of definite therapeutic advantage to the patients of younger age group.

In cases of burns, a marked haemolysis was evident during the first three weeks of the injury. In all cases, a mild degree of splenic sequestration was observed. No evidence auto-immune phenomenon was observed even on followup of the cases upto a period of 12 weeks.

Radio-ferrokinetic :

Since the introduction of radio-iron tracer techniques, a new dimension in the investigations of haematological disorders has been available. Like other tracer techniques, this provides us with a method for studying the dynamic events of haemopoesis. The most effective use of radio-ferrokinetic has been found in the study of the cell kinetics of bone-marrow. Anaemia is the result of haemopoetic disorder, leading to arrest of erythropoetic maturation, be it due to nutritional deficiency or bone-marrow failure, due to primary or secondary causes. Our primary aim, in the present study was to have a comprehensive evaluation of radio-ferrokinetic as a clinical investigation tool in cases of anaemia. The study was therefore carried out in anaema of known and unknown aetiology.

The results in well established cases of iron deficiency (total 85) clearly showed that a distinctive pattern of short plasma iron disapperance half time (PIDT½) with increased plasma iron turn over rate (PITR) is diagnostic of a state of hypoferraemia with proliferative erythroblastic bone-marrow. Similar results may however be seen in cases of haemolytic states, (Table 6,7), but in such cases an estimation of serum iron and bone-marrow haemosiderin could eliminate the state of hypoferraemia.

A paradoxical result of short plasma iron disappearance half time, increased plasma iron turn over rate with a normal to increased serum iron level has however been observed in cases of malignancy. These results corroborate our observations published earlier (Banerjee and Narang, 1967). The explanation of this phenomenon has been suggested to be of an extra medullary iron shunt to spleen and liver.

In conditions of bone-marrow inadequacy either due to primary or secondary hypoplasia, radioferrokinetic results are diagnostic of non-utilisation of administred iron by the haemopoetic system. In our series, the study has been carried out in primary aplastic anaemia, and in drug-induced hypoplastic anaemia due to Chlormycetin, Novalgin, Butazolidin, and Neomarcazol therapy. The results have shown a poor avidity of bone-marrow for iron, in unduly prolonged PIDT½, low PITR and poor ^{59}Fe localisation in the bone-marrow. In some cases of primary aplastic anaemia, however, evidence of extra medullary iron localisation in spleen and liver was observed. Evidence of extra medullary haemopoesis was observed to a conspicuous feature in cases of Myelosclerosis. All the cases of myelosclerosis presented splenomegaly and the results of radio-ferrokinetics suggested contraindication of splenectomy in such cases.

In myelopathisic anaemia of lukaemia, lymphoma or metastatic carcinoma, the results of radioferrokinetics were similar to that of hypoplastic anaemia.

The evaluatoin of the results of radio-ferrokinetic therefore showed that as a clinical investigative procedure its major role lies in differentiating between an anaemia of hypoplastic or functionally inadequate bone-marrow and an anaemia of hyperreactive bone-marrow either of nutritional or haemolytic aetiology.

Simultaneous Radio-Erythro-ferrokinetic :

In haematological disorders presenting normocytic hypochromic anaemia (secondary) there is often an interplay of more than one actiological factors. This presents basic problems in the management of such anaemia. Classical examples of such problems are anaemia of malignancy, portal cirrhosis, renal failure or anaemia of burns. While a degree of deficiency-anaemia due to chronic blood loss in such conditions is well known, because of the other aetiological factors such as erythroblastic maturation arrest or latent haemolysis, anaemia often proves refractory to the treatment with iron therapy. In such cases therefore it becomes necessary to do a comprehensive radio-erythro-ferrokinetic studies. Such studies are helpful in detecting the multifactorial aetiology.

Procedure wise, as we have already seen, each of the investigation of erythrokinetic and ferrokinetic are time consuming, extending over weeks. It is therefore necessary to evolve a method by which both the studies can be carried out simultaneously in the same patient. Technically this is feasible because of the distinctive physical properties of the radiation emitted by ^{51}Cr, a predominent gamma emitter (o.237 Mev) and radioactive iron ^{59}Fe, a beta and gamma emitter. The radioactivity of the two radioisotope in the same sample can be conveniently differentiatiated by a scintillation counter equipped with a pulse height discriminator.

In the present study the simultaneous study was carried out in 323 cases. The results (Table-7) have shown that a simultaneous erythro-ferrokinetic study is useful in diagnosing the multiple aetiological factors such as post haemorrhagic iron deficiency, latent haemolytic state and bone-marrow inadequacy. The procedure is also convenient and economic for a significant reduction in the total investigative period of the patient.

The advantages of simultaneous radio-erythro-ferrokinetic has been convincingly brough out in cases of burns, where the major cause of anaemia in the first week was found to be post-haemorrhagic and haemolytic, while after 12 weeks, the anaemia is predominantly due to a state of hypoplastic bone-marrow. Similarly in cases of chronic renal failure a latent haemolytic state with a hypoplastic bone-marrow appeared to be the major causes of anaemia.

CONCLUSIONS

1 An evaluation of the radio–erythro–ferrokinetic studies has been carried out in 477 patients presenting normocytic hypochromic anaemia of prolonged morbidlity. The present study is one of the most extensive report published so far and first of its kind in the country.

2 The study has convincingly established the diagnostic value of radio–erythro–kinetic with labelled autologous and homologous red cells in haemolytic anaemia of primary, secondary, corpuscular, plasma factors and of splenic aetiology.

3 The study has convincingly established the diagnostic value of radio-ferrokinetic in anaemias of dyshaemopoetic disorder.

4 The study has shown the important prognostic values of both the radio–kinetic and radio–ferrokinetics studies in deciding the specific therapeutic approach, medical or surgical in normoytic anaemia, refractory to routine haematinic therapy, as well as in assessing the therapeutic response to treatment.

5 The study has covincingly established the investigative or research value of the radio–erythrokinetic and radio–ferrokinetic in the understanding of the pathogenesis of secondary anaemia accompanying systemic disorders.

6 The work has confirmed the technical and economic advantages of a simultaneous radio-erythro-ferrokinetic in obtaining simultaneous information about the possible haemolytic and dyshaemopoetic aetiologies. Besides, the technique considerably reduces the investigative period to about four weeks.

7 The study has convincingly established the scientific advantages of the radio-erythro-ferrokinetic studies in arriving at an early diagnosis and management decision, thereby reducing the indefinite period of morbidity and prolonged hospitalisation of such patients.

8 Radio-erythro-ferrokinetic studies are therefore considered to be important nuclear methodologies, in the management of chronic normocytic anaemia and deserve to be introduced in the large referral hospitals for technical as well as economic reasons.

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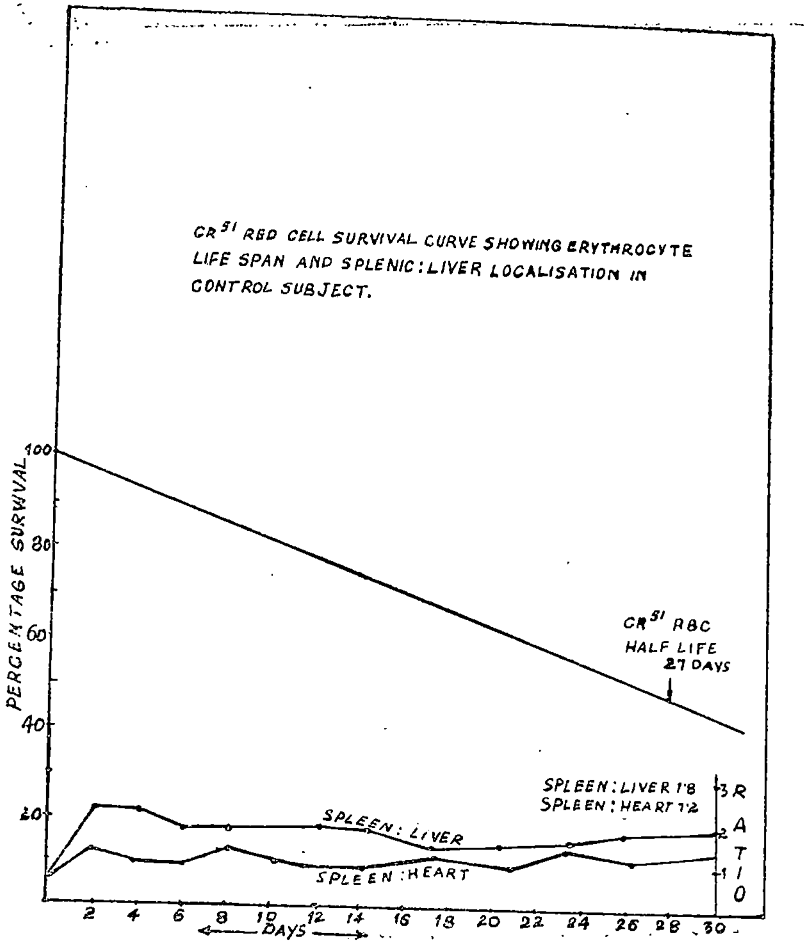

Fig. 1

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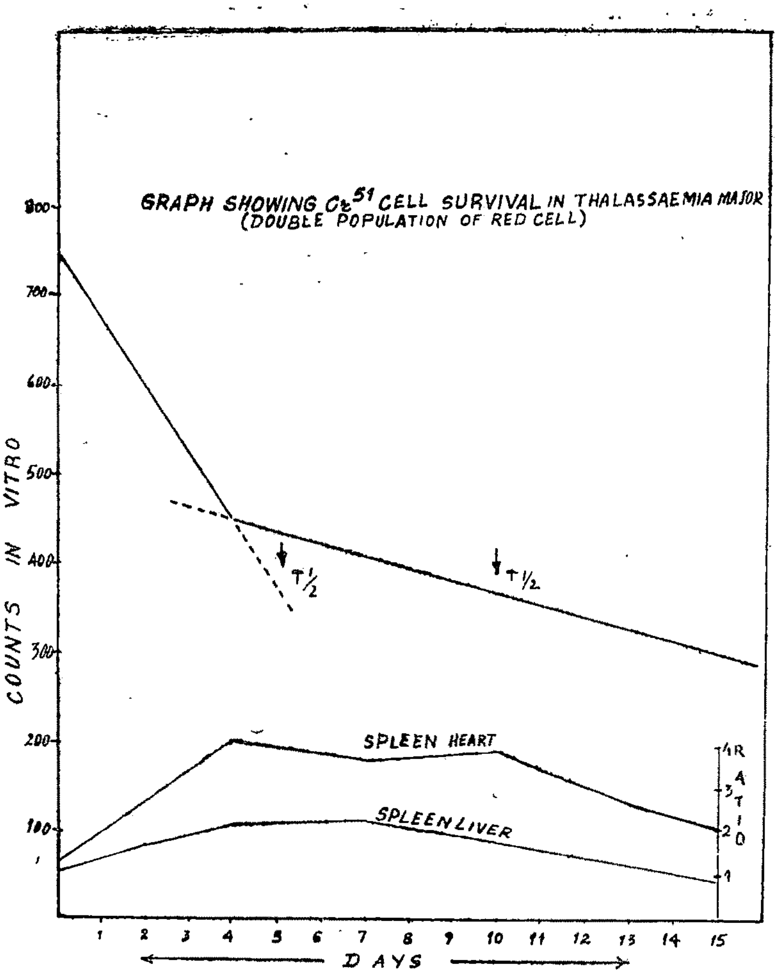
GRAPH SHOWING Cr51 CELL SURVIVAL IN THALASSAEMIA MAJOR
(DOUBLE POPULATION OF RED CELL)
800
700
600
500
400
300
200
100
COUNTS IN VITRO
T½
T½
SPLEEN HEART
SPLEEN LIVER
4
3
2
1
RATIO
1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
DAYS

Fig. 2

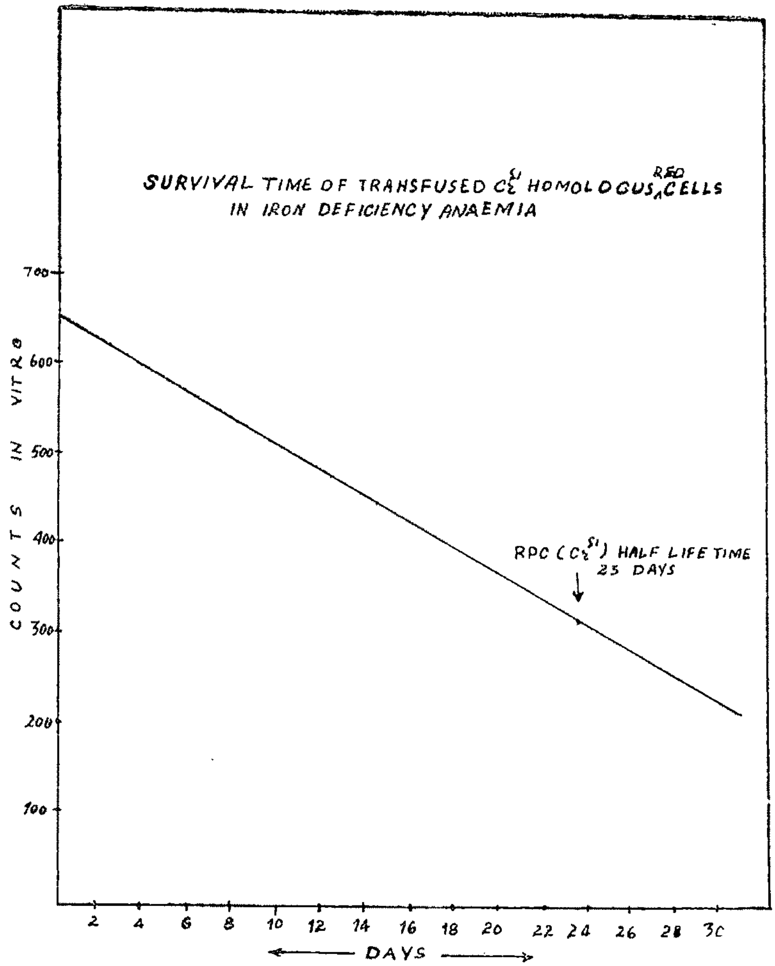
SURVIVAL TIME OF TRANSFUSED Cr^{51} HOMOLOGUS RED CELLS
IN IRON DEFICIENCY ANAEMIA
COUNTS IN VITRO
700
600
500
400
300
200
100
RPC (Cr^{51}) HALF LIFE TIME
25 DAYS
2
4
6
8
10
12
14
16
18
20
22
24
26
28
30
DAYS

Fig. 3

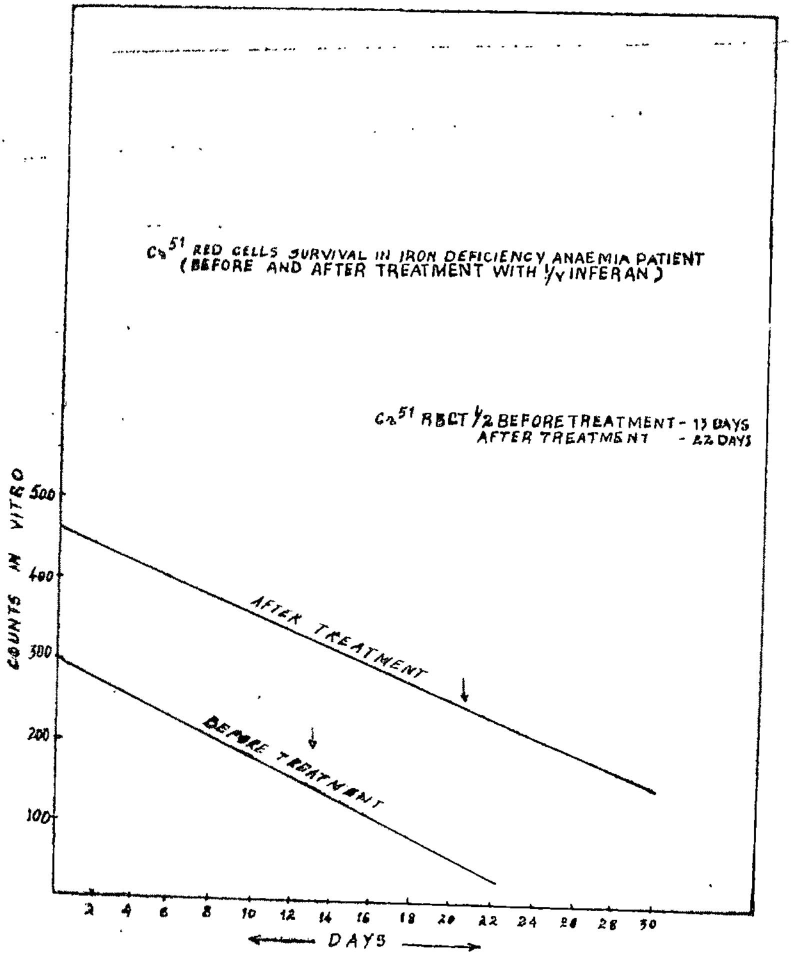
Cr^{51} RED CELLS SURVIVAL IN IRON DEFICIENCY ANAEMIA PATIENT
(BEFORE AND AFTER TREATMENT WITH I/V INFERAN)
Cr^{51} RBCT ½ BEFORE TREATMENT - 13 DAYS
AFTER TREATMENT - 22 DAYS
COUNTS IN VITRO
500
400
300
200
100
AFTER TREATMENT
BEFORE TREATMENT
2
4
6
8
10
12
14
16
18
20
22
24
26
28
30
DAYS

Fig. 4

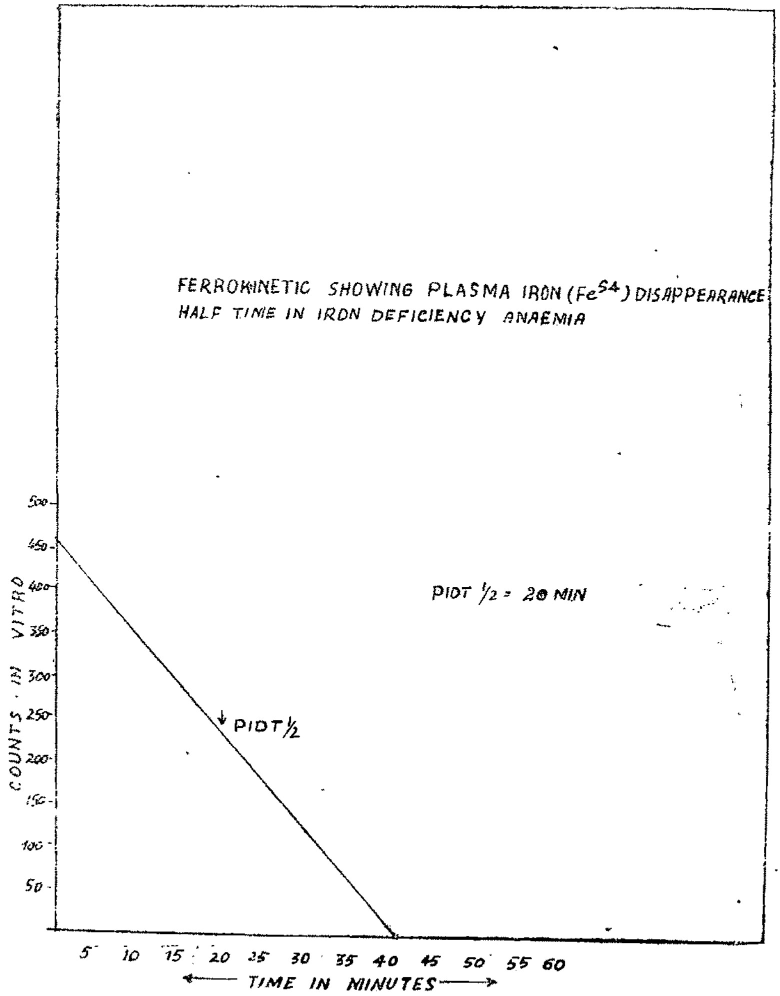
FERROKINETIC SHOWING PLASMA IRON (Fe54) DISAPPEARANCE HALF TIME IN IRON DEFICIENCY ANAEMIA
PIDT ½ = 20 MIN
PIDT½
COUNTS IN VITRO
500
450
400
350
300
250
200
150
100
50
5
10
15
20
25
30
35
40
45
50
55
60
TIME IN MINUTES

Fig. 5

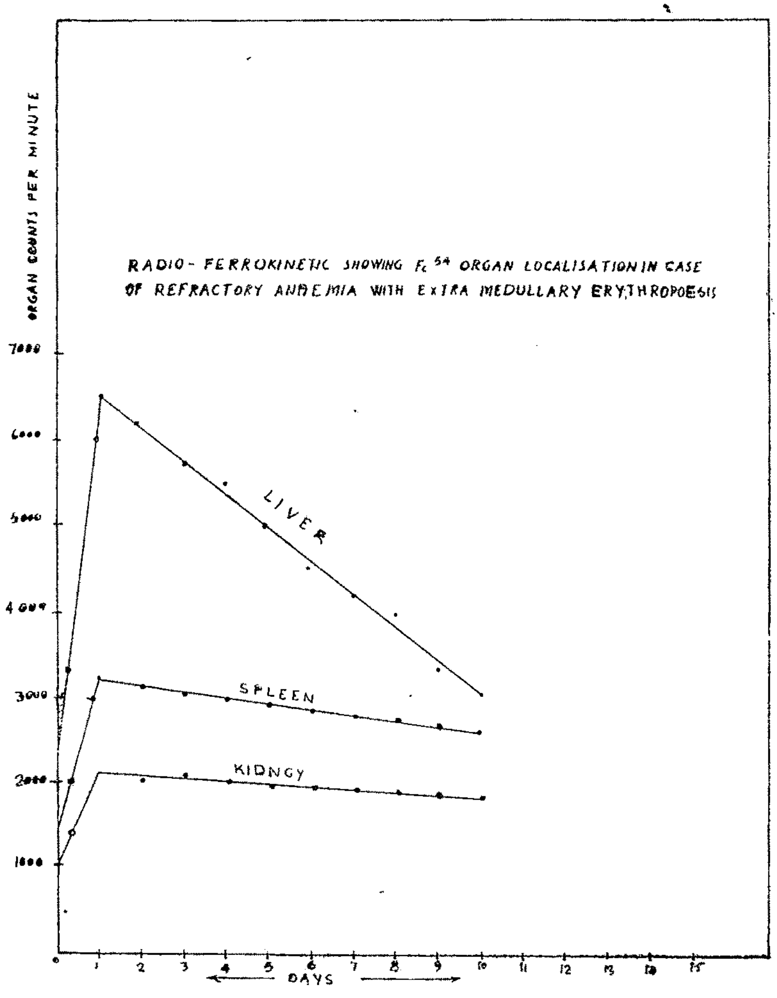
RADIO- FERROKINETIC SHOWING Fe^{54} ORGAN LOCALISATION IN CASE
OF REFRACTORY ANAEMIA WITH EXTRA MEDULLARY ERYTHROPOESIS
ORGAN COUNTS PER MINUTE
7000
6000
5000
4000
3000
2000
1000
LIVER
SPLEEN
KIDNEY
0
1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
DAYS

Fig. 6

the Medical Superintendent of Safdarjang Hospital for their kind permission to carry out the research work.

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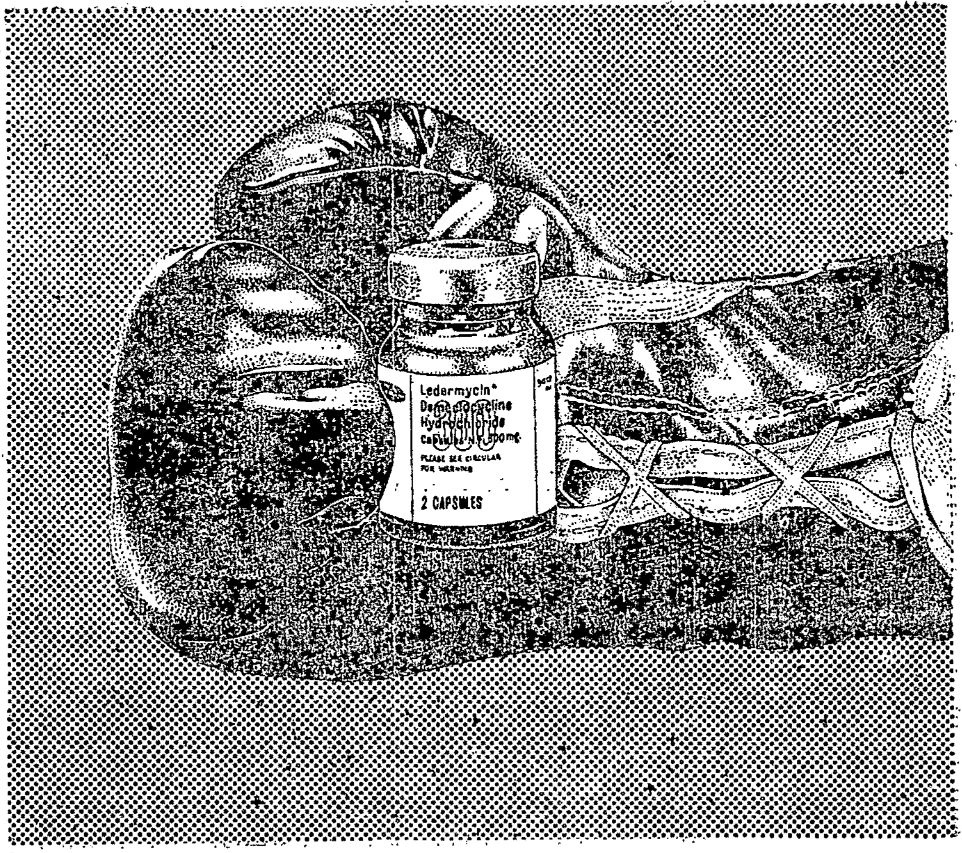

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Soluble Tablets: 30 mg., Strip of 10.

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LORD'S PROCEDURE AND REGIME FOR THIRD DEGREE HAEMORRHOIDS

(A simple Out-Patient treatment for Haemorrhoids)

BY

Major T.K. Cherian*

INTRODUCTION

Lord's procedure for haemorrhoids (LPH) introduced in 1965 by Mr. Peter H. Lord, Consultant Surgeon, Wycombe General Hospital, High Wycombe, United Kingdom is a new concept in the treatment of a condition, in which the very thought of haemorrhoidectomy and the pain and discomfort thereafter, prevents many a patient from seeking medical advice early. This fearful attitude of the patient worsens the condition and leads to delay in cure. It is in this context that the simple, relatively harmless method of L.P.H. becomes a valuable addition to the armamentarium of the Surgeon. No doubt, it cannot be considered a 'panacea' for all cases of haemorrhoids ; but the follow up of over 1500 cases done in the Wycombe group of hospitals during the past 7 to 8 years has shown that the procedure and regime is curative in the vast majority of cases. Similar results have been recorded by MacIntyre and Balfour (1972), Fussel (1973) and Sames (1973).

The incidence of haemorrhoids and other benign anorectal diseases are significantly high in the Armed Forces, due to obvious predisposing factors like irregular timings of meals and incongenial toilet facilities especially in the field.

On an average, haemorrhoidectomy involves hospitilization of the patient for 2 to 3 weeks with associated loss of man power and hospital resources. From the patient's angle, the post-operative pain and discomfort, particularly the agony of the first bowel movement after the operation, are something dreadful and never forgotten. These being so, L.P.H. opens a new approach towards the management of a condition which is estimated to afflict about 40% of the population at one time or the other.

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MATERIAL AND METHOD

Cases of haemorrhoids treated by L.P.H. and regime in High Wycombe group of Hospitals from 1965 to July 1973 have been followed up for a month to 7 years. Recent cases were personally followed up by me during my stay in Wycombe General Hospital from May 1972 to August 1973, while the old ones were reviewed from the hospital records.

Table 1

Year	Number of 3rd degree Haemorrhoids treated by L.P.H. and Regime in High Wycombe Group Hospitals
1965–1968	345 (approximate)
1969	224
1970	263
1971	275
1972	258
Up to July 1973	147
Total	1512

Method of Treatment :

The treatment consists of two parts.

(a) L.P.H. – Manual dilatation of anal canal under a short general anaesthesia done as an out–patient procedure in the operating theatre.

(b) Regime – A follow up regime of periodic dilatation carried out by the patient at home using Lord's pattern anal dilator, (Plate No. 1) according to a specified schedule (see below).

Theoretical Basis of Treatment :

According to Lord (1968), haemorrhoids are reversible and are caused by narrowing of the anal canal and lower rectum by constricting band(s), which in turn produce varicosity of haemorrhoidal plexus of veins. Hence he tries to tackle the cause of haemorrhoids by his dilatation procedure.

The narrowing of anal canal was first documented by Ernest Miles in 1919. He described the 'pecten' band as a rubber umbrella ring and thought that it was produced by the passive congestion of tissues due to varicosity of the haemorrhoidal veins (haemorrhoids) which ultimately leads to fibrous induration. This band is purely pathological and does not exist in healthy anal canal. It can be of varying thickness and density, completely encircling the lower part of the anal canal and is situated between the mucous membrane of the pecten and the external sphincter. In well marked instances, the band of fibrous tissue can be felt distinctly by the examining fingers. Miles thought the 'pecten' band was the result of haemorrhoids. But Lord (1969) belives that the constriction band to be the cause and that the band is not always at the level of the pecten. According to him there can be more than one band which can be superficially or deeply placed in anal canal and lower rectum and the 'pecten band' as described by Miles is only one example of the several constricting bands found in this region. Lord now thinks that these constrictions are analogous to the constrictions at the recto-sigmoid junction seen in diverticular disease (personal communication.)

In support of this theory, he postulates that

(i) in the natural history of the haemorrhoids there are always periods of remission of symptoms. An attack of bleeding is brought on by constipation. If bowel action does not happen, the patient is virtually free of symptoms; but they return with the passage of hard stools next time. This shows that the condition is precipitated by passage of hard stools through a narrowed anal canal and that the condition reverses itself when the stools are soft and easily passable.

(ii) if a patient is proctoscoped in between the attacks, after he lies down for an hour or so, the haemorrhoids would be empty and the findings would be normal at the introduction of the proctoscope, but they can be made to reappear by partially withdrawing the scope and pressing it against the anal ring (which is done often during the injection treatment of haemorrhoids).

(iii) he believes that there are two characteristic symptoms in nearly all cases of internal piles, attributable to the constricting band(s), apart from those due to piles themselves. They are (a) difficulty in complete emptying of the rectum during defaecation and (b) the diminished size and calibre of stools passed; the motion being voided in short pieces about the size of an index finger.

Thus Lord (1972) puts forth the following hypothesis to explain why haemorrhoids occur, why they are related to defaecation and why they are cured by his procedure and regime.

In normal defaecation, the anal sphincter mechanism relaxes and dilates to allow easy passage of faecal mass. Even when this mass is of very firm consistency, no marked rise in the intraluminal rectal pressure is required for its expulsion. If, however, the anal ring is unable to dilate owing to some form of fibrous constriction, defaecation must be accompanied by a rise in intra-rectal pressure. This rise in intra-rectal pressure has only to exceed the venous pressure to cause congestion of haemorrhoidal plexus. The resultant swelling will further restrict the already narrowed outlet. If the band(s), are destroyed by stretching, the vicious cycle can be broken; rectal pressure thus returns to normal and haemorrhoids resolve. These facts are well borne out by the dramatic results of Lord's procedure.

DIAGNOSIS AND PREOPERATIVE MANAGEMENT

Though haemorrhoids are classified into 3 degrees, there is no sharp demarkation between the degrees. Third degree haemorrhoids are defined as those which prolpase at stool, and have to be pushed back by the patient. This is a matter of report and the diagnosis is made mainly on the history and proctoscopic findings. Other lesions should be excluded by sigmoidoscopy.

The patient is seen in the Out-Patient Department and a date arranged for his treatment as an out-patient (Day case). On the day of the operation, he comes prepared for a general anaesthesia.

The instruments required are shown in plates No. 2 and 3.

Though instruments are sterilised, no attempt is made to carry out the procedure aseptically, Instruments consist of :—

Proctoscope
Sigmoidoscope
Battery Box
Biopsy forceps
Probe
A dish of Hibitane or Cetavlon
A number of polyvinyl sponges 15 cm x 10 cm x 5 cm
Sponge holding forceps
Lord's Pattern Perianal skin tag forceps
½" Duval's tissue forceps
Scalpel
Scissors
Zn Oxide strappings
Swabs, dressing and a T. bandage.

The Procedure :–

The patient is asked to lie on his left side with his knees drawn up. The anus is inspected with a good shadowless lamp.

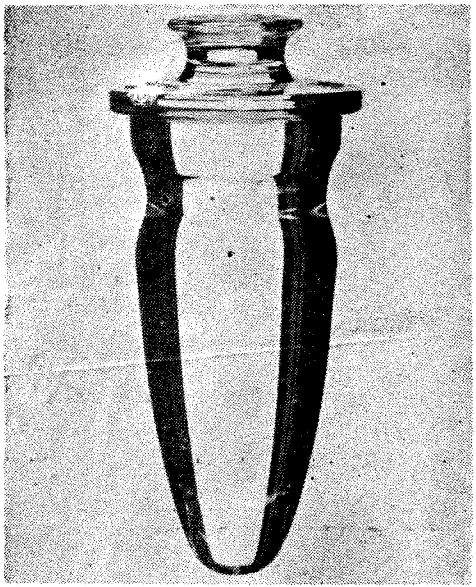

Fig. 1 Anal Dilator (Lord's Pattern Mark 2)

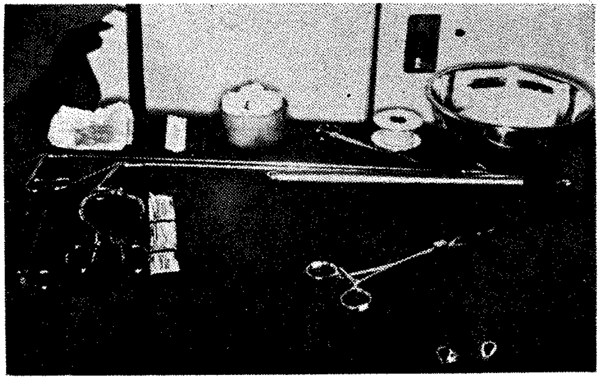

Fig. 2 Lay out of the trolley for L.P.H.

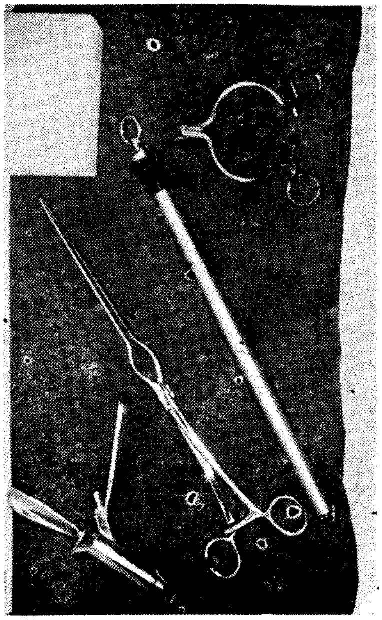

Fig. 3 Instruments for LPH :–
L to R – Proctoscope, Sponge introducing forceps, Sigmoidoscope ; Lord Pattern skin tag Clamp, and Polyvinyl Sponge

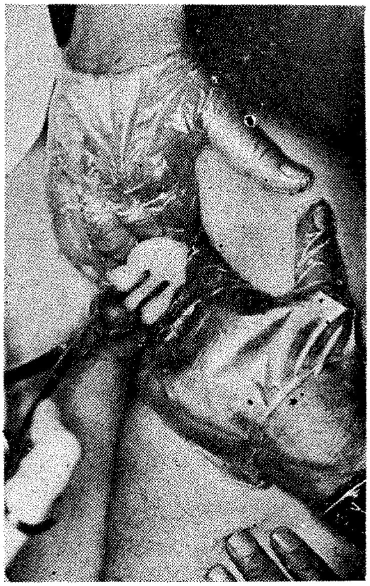

Fig. 4 L.P.H.–The sponge being introduced after dilatation

The anaesthetic consists of an I. V. injection of Epontal, supplimented if necessary, by Halothane. If the outpatient proctoscopy and sigmoidoscopy were not satisfactory, they are repeated. The procedure is then carried out and it takes less than a minute in most cases. The constricting band can be felt by the index fingers of both hands in the rectum pulling the rectal walls in opposite directions. It cannot be felt with one finger alone (Lord 1972). The band can be very thin and narrow and submucous or it can be felt as part of the sphincter. The higher bands when present, can only be felt after dilating the lower ones. Once the bands are felt, they should be gradually stretched manually until they give way. This is sometimes easy, but can involve a considerable force. The dilatation should extend above the anal canal and include the lower rectum. It is best to avoid strain at 6 and 12 O'clock positions as these are the weak spots of the sphincter mechanism. The main effect of dilatation should be aimed laterally at 3 and 9 O'clock positions. It is convenient for the surgeon to stand against the patient with his back to the head of the patient. Tearing must be avoided and this can be done by gentleness in the dilatation which is best combined with rotatory movements of the fingers. This spreads the dilatation uniformly in all the four quadrants of the orifice. It is unlikely that an instrument would do this as well as the surgeon's fingers, as the surgeon soon learns to feel when the bands have given way and learns to avoid tearing. As with any other surgical procedure, better results are obtained with experience. The extent of dilatation will vary up to 8 fingers, so often advised ; but it is up to the surgeon to decide how far he has to stretch in a particular case. If he is satisfied with a 5 to 6 finger dilatation, it would be unsafe to do more. It is always better to do little than too much.

As soon as dilatation is achieved, a moistened sponge held in a sponge holding forceps (specially designed by Lord) is thrust into the lower rectum and anus while the fingers are still in postition. The sponge is left in situ for an hour which reduces the chances of a haematoma developing (Plate No 4).

Post-operative Care

The patient quickly recovers from the anaesthetic and receives an analgesic by injection, if needed. Most patients do not require this. The sponge is removed after an hour by a nurse and it seldom causes pain or difficulty. By this time the patient should be sitting up and soon be ready to be taken home ; where he is advised to move about as much as he can and to report to the hospital immediately if there is any increase of pain or swelling. He is otherwise seen in the Out-Patient Department after two weeks.

Meanwhile he starts the regime according to instructions given. He is provided with a dilator (Lord's Pattern) and a tube of lubricating jelly. (K. Y. Jelly). He is also given Normacol and a card with instructions. The card explains to the patient that the regime has two objectives. Firstly, the regular use of anal dilators stops the recurrence of anal narrowing during the period of healing. Secondly the use of Normacol ensures bulky soft stools which help restore regular bowel habits and prevents local trauma which could be caused by passage of scybullae.

The well lubricated dilator should be passed slowly to its full extent and should be left in situ for a minute and then removed slowly. This is to be done each evening for two weeks ; every other evening for the following 2 weeks and then once a fortnight for a total duration of 6 months, by which time the anus should be fully healed with a normal lumen. The dilator is left with the patient and he is advised to use it any time he has anal symptoms.

The dilator has been specially designed for this regime with a maximum diameter of 3.8cm. The nose is blunt. The effective part is the middle of its body and it has a gentle taper followed by a groove and a handle so that the patient knows when it has gone in all the way.

Normacol dosage (usually 5 ml. at bed time and in the morning) is adjusted by trial and error, taken with plenty of fluids. Once regular bowel habits have been established, Normacol is gradually reduced till it is no longer required.

Follow up and Complications

Most of the patients seen 2 weeks after L.P.H. report that there is no bleeding, no prolapse and that they have full control of flatus and faeces with regular movements of the bowel. These patients are discharged from the Out-Patient Clinic with instruction to return if they have further trouble. Some require further follow up and treatment.

The following are some of the complications that may occur :-

(a) **Incontinence** - This is the most important complication, to be avoided. Even a small number of cases developing incontinence after L.P.H. would totally discredit this method of treatment. Permanent incontinence has not happened in this series; but minor degrees of temporary incontinence do occur; particularly where this method was emplowed in conditions other than third degree haemorrhoids. It works well in fissure-in-ano and is a useful adjunct to fistulectomy and it is very tempting to use it for fibrous stricture of anus with pain. In these conditions, a higher incidence of incontinence should be expected. It the procedure is strictly reserved for haemorrhoids incontinence is surprisingly rare. This method can be safely used in all age groups (Lord 1972), but according to Hawley (1973) it is better avoided in elderly patients with lax anuses and in those with a short anal canal and wide anorectal angles, where it can precipitate prolapse of rectum. Mac Intyre and Balfour (1973) also found incontinence a minor problem in their series with 21.8% incontinent for flatus but only 3.6% incontinent for faeces. Fussel (1973) noted incontinence in 20% of cases.

There is much difference of opinion as to what constitutes incontinence and hence wide variations in the percentage of cases developing this complication are possible. Some surgeons disregard all minor soiling episodes and consider the patient incontinent only if a full bowel evacuation occurs without the patinet's awareness; while others regard even a little soiling or incontinence of flatus as evidence of incontinence. Lord states that initially patients may have an occasional episode of incontinence but that will rapidly return to normal. .If there is any problem the patient can be taught pelvic exercises. It is important to understand which of the possible machanisms is at fault. In most cases, it is merely a matter of re-education. The patient who has had a real problem for years to expel the rectal contents through a narrowed passage, suddenly finds himself faced with the. problem of holding them; particularly flatus. The automatic function of his sphincter has been impaired through years of straining at stools. Also the local bruising caused by L.P.H. numbs the area and interferes with the sensory function, temporarily. All these contribute to a temporary lack of control of his sphincters, which can be regained by reeducation of exercising the sphincter muscles. In troublesome cases faradic stimulation of the sphincter is useful; but it is seldom required. Overall, the post-operative problem of lack of control of flatus or faecal soiling is comparable with that following haemorrhoidectomy. Golighen at al (1969) reported an incidence of 20% incontinence of faeces and flatus three months after a four finger dilatation of anal sphincter during haemorrhoidectomy.

(b) **Bruising and Haematoma**:- A certain amount of bruising without haematoma is normal following L.P.H. If swelling does occur – which is seldom – the patient should be hospitalised till it subsides. It does not give rise to any serious consequences and this is the experience of all surgeons practising this mothod.

(c) **Splitting**:- The anal skin tends to split and crack during L.P.H. Minor fissuring is of no consequence, but when deep fissuring occurs (which should of course be avoided), hospitilisation is advisable to ensure that it does not get infected.

(d) **Prolapse**:- Mucosal prolapse is a worrying problem in about 10-12% in this series requiring removal of mucosa with special clamps.

MacIntyre and Balfour did not face this problem; but they found that skin tags require excision.

Lord excises skin and mucosal tags under General Anaesthetic using his special crushing clamps. The skin tag is grasped with a Duval's forceps and the clamp is applied across its base. The clamp is then closed tightly and strapped to the buttocks and the redundant mucosa, is shaved off with a knife. The same procedure is applicable to mucosal prolapse and sentinel piles. The clamp is removed after one hour usually without any bleeding. If clamp excision is combined with L.P.H. the sponge is removed at least ten minutes before the clamp is removed; to avoid bleeding from the site excision. Complete rectal prolapse has not been reported so far.

(e) **Bleeding:–** Post operative bleeding has not been a problem even when clamp excision is employed. To date serious haemorrhage requiring surgical intervention has not been reported in any of the series. The procedure has been so good in stopping the bleeding of haemorrhoids that, if any bleeding persists after a few weeks postoperatively it is important to look for other causes.

(f) **Strangulated Haemorrhoids:–** Though this method works dramatically in patients with strangulated haemorrhoids, these cases should be approached with extreme caution as the operative field is infected and the danger of ascending infection is ever present.

RESULTS

L.P.H. has been carried out in these groups since 1965 and so far the results have been very encouraging. This is borne out by the fact that haemorrhoidectomy operation has gone down from an average of 50 a year to about 5 a year, though the number of cases of haemorrhoids treated has gone up considerably. More and more haemorrhoid cases are seeking treatment early now and the total number of surgical cases has more than doubled between 1964 and 1973.

Table II

Year	No. of Surgical Cases at Wycombe General Hospital	Haemorrhoidectomy
1961–1964	2850 (average)	54 (average)
1965	3364	52
1966	3411	38
1967	3955	9
1968	3725	6
1969	4290	6
1970	4381	3
1971	5854	3
1972	6132	4

DISCUSSION

So far there is nothing to suggest that the long term results are going to be any less satisfactory than the immediate results, but it will be several years before this method is fully tested. In a small number of cases the end results have not been satisfactory and they were treated with formal haemorrhoidectomy. Even in these, nothing was lost by the dilatation. MacIntyre and Balfour (1972) showed a success rate of L.P.H. comparable to that of haemorrhoidectomy. Fussel (1973) has reported 80% success in curing haemorrhoids by L.P.H., Sames (1973) also reported 80% success.

Hood and Williams (1971) compared this method wtih Barron's rubber band technique and they found only 30% success with L.P.H.; but they have not been using the dilator and they have been restricting the dilatation to only four fingers. Hawley (1973) found this method useful in prolapsed and strangulated haemorrhoids. He also found relief of pain and bleeding of all haemorrhoid cases he treated by this method; but found that internal haemorrhoids can still persist and result in prolapse, dampness and irritation. The number of patients and degree of dilatation differed in the above series from that advocated by Lord.

Currently L.P.H. is being accepted world wide and there are many centres in the United Kingdom (Edinburgh, Oxford, Winchester, Bath, Dublin, Wigan) where it is considered as the initial treatment for third degree haemorrhoids. Carden (Australia), Lannon (Johannesberg, South Africa), Georgulius (Athens, Greece), Eddy (New York, U.S.A.) are some of the other surgeons using this method at present.

Because of the simplicity of the procedure, general practitioners have been referring first and second degree haemorrhoids also for L.P.H. and the results so far have been encouraging. Those haemorrhoids which are cured by injection treatment, do equally well with L.P.H. In fact such cases may benefit from this method of treatment by way of removal of the cause for the condition. In that context L.P.H. may be considered as a preventive measure. Further follow up of first and second degree haemorrhoids will be required to assess this aspect of the L.P.H. However this appears to be superior to injection therapy as it avoids the fibrosis of the injection therapy which can precipitate the band formation.

SUMMARY

The dilatation procedure and regime of Lord can be safely advocated for treatment of third degree haemorrhoids. They are aimed at the cause of haemorrhoids, which according to Lord is the narrowing of the anal canal by constricting band(s) during the healing stage. In Recent Advances of Surgery (1973) edited by Selwyn Taylor, Hawley states, "without doubt this procedure (LPH) is an interesting concept and may find a permanent place in treatment of haemorrhoids. It is too soon to evaluate the late results

and to know the eventual recurrence rate ——— While the possibility of producing an incontinent patient or a complete rectal prolapse must always be borne in mind, experience suggests that these complications can be avoided by careful patient selection."

ACKNOWLEDGEMENTS

My sincere thanks are due to Mr P.H. Lord for allowing me to practice this method of treatment on some of his patients and to follow up all his cases and also for the advice and encouragement he gave me in preparing this paper. I wish to thank the High Wycombe and District Hospital Management Committee (UK) for permitting me to review the case records.

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AUSTRALIA ANTIGEN AND PROTECTIVE INNOCULATION- A STUDY IN TRAINED SOLDIERS AND ARMY RECRUITS

BY

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ABSTRACT

929 trained armed forces personnel were tested for HBAg and their result was compared with that of 363 army recruits. The incidence of HBAg in trained soldiers was found to be significantly at a higher level than that in recruits. This incidence in the trained soldiers was compared with the incidence of HBAg in 966 healthy civilian blood donors. There was no statistical difference between these two groups of people.

Reasons for this higher incidence of HBAg in the trained soldiers as compared to army recruits have been discussed in this article.

INTRODUCTION

Blumberg and his associates discovered in 1965 a new antigen called Australia antigen (HBAg) which subsequently was found to be associated with serum hepatitis (Prince, 1968; Krugman and Giles, 1970). Present evidence shows that there are two types of Viral hepatitis, one due to Virus A and the other, due to Virus B. (MacCallum and Bradley 1944; Neefe et al, 1945 ; Krugman and Giles, 1972). Virus A hepatitis is primarily an enteric infection with short incubation period (15 to 40 days), viraemia and fecal excretion of virus. Mode of transmission is by faecal–oral route. Virus B hepatitis is characterised by long incubation period (50 to 180 days) caused by parenteral infection. At first it was thought that the Virus B infection spreads only by inoculation but recent studies (Krugman & Giles 1972) have shown that it can be acquired by contact as well as by the parenteral route. Virus B hepatitis is more dangerous of the two, as fulminant viral hepatitis, chronic active or aggressive hepatitis and chronic liver disease progressing to subacute hepatic necrosis and post necrotic scarring are always related to infection with hepatitis virus B (Alter, 1972, Boyer 1972; Redeker 1972). Earlier studies by Dutta and Saha (1973) showed a high incidence of 2.88 percent HBAg positive sera amongst 727 Armed Forces personnel. As the reason for this high prevalence of Australia antigen in apparently healthy soldiers was not understood in the above study, the present authors therefore extended their study, amongst Army recruits to find out the prevalence rate of HBAg amongst them.

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The most obvious differences in acquiring Hepatitis B virus infection between these two groups is that the trained soldiers as a rule get their annual innoculations and bi–annual vaccinations whilst the recruits in general, had their innoculations and vaccinations for the first time in their life when they reported to the training centres. Secondly, the trained soldiers, depending on their years of service, have been living in the army barracks as a tightly–knit community for a fairly long period.

Present paper is a study on the level of incidence of Au-antigen (HBAg) in the trained Armed Forces personnel vis-a-vis raw recruits of the army and an analysis of causes for the high incidence of HBAg in the first group.

MATERIAL AND METHOD

Material :–

(a) 929 trained Armed Forces personnel drawn from various local units were tested for HBAg. Most of them had more than 6 to 7 years of service. Some of them had more than 10 years and a few more than 20 years.

(b) 363 raw recruits drawn from a local regimental centre were tested for HBAg. They all belonged to one particular class and are recruited from villages of three neighbouring states. Practically none of them had any injections earlier in their village life, and all were given TAB, T.T. and vaccination within a week after their arrival in the centre. Second dose of TAB was given after a week and T.T. after six months. Vaccination was repeated only after 7 days if not successful at the first time. Blood for HBAg testing was collected within 6 months to one year of their arrival in the centre.

(c) 966 civilian healthy blood donors coming from various walks of life tested for HBAg have also been included in this study.

(d) 152 leprosy sera were obtained from 2 leprosy Centres, one at Agra (Army Hosp.) and the other at Sahdra (by courtesy of Dr. N. M. Chawla).

Method

Blood collected from these personnel was allowed to clot. Sera, thus seperated were put in 1 ml glass ampoules, sealed, labelled and stored at–20°C till they were brought out and tested for HBAg. The method of detection employed was that of Dutta and Mohammed (1972). Australia antibody (anti HBAg) was obtained from multiple transfused cases and was confirmed against standard sera kindly supplied by Prof. J.P. Soulier of Centre National de Transfusion Sanguine; Paris.

RESULTS & DISCUSSION

Table 1 below shows the incidence of HBAg and anti HBAg in trained Armed Forces personnel and recruits.

Category of Donors	Total No. Tested	Positive for HBAg		Positive for anti HBAg	
		Number	Percentage	Number	Percentage
Trained Soldiers	929	24 p=	2.6 0.0258	4 p=	0.43 0.0110
Recruits	363	4	1.1	–	–
	P<0.05				

Most of the trained armed forces personnel had more than 5 years of service. Their HBAg incidence was found to be 2.6 percent and that of recruits 1.1 percent.

Statistical analyses showed a significant level of incidence ($p<0.05$) of HBAg in the trained soldiers as compared to the recruits. Basic differences between these two groups of people are : (a) a community–living in case of trained soldiers for a long period and (b) periodic innoculation. Majority of the recruits had their first innoculation when they reported to their training centre. Further, they were living in their villages and had individualistic life. Both these two factors could lead to an increased incidence of HBAg especially the second factor. Present system of giving annual innoculations and bi–annual vaccinations to a large number of soldiers on the same day at the same place at the same time may have contributed to this higher incidence of HBAg in trained soldiers. It is now known that communality of the agent is responsible for most cases of viral hepatitis. This has further been substantiated by subtyping (Mosley 1972). It has been found by Nielsen etal (1973) that Y (ay+d–) sub type was more frequent amongt drug users (98 percent) and also in case of acute viral hepatitis (79 percent). Whilst D (ad+y–) was the predominant one amongst blood donors.

It is now known that anicteric carriers often progress to subacute hepatic necrosis and post necrotic cirrhosis (Klatskin, 1958). Similarly, Sherlock et al (1970), Prince (1958) have also presented cases of HBAg positive chronic liver disease without any previous history of acute viral hepatitis. Therefore, such anicteric asympytomatic carriers of HBAg in the Armed Forces personnel should be followed up regularly. Further, a single innoculation through separate autoclaved needles to each individual in the M.I. Room and giving to fewer soldiers on different days and at different times may bring down this high HBAg incidence. In order to elucidate further this high incidence level of HBAg in the trained soldier, we carried out a survey of HBAg amongst civilian healthy blood donors at Delhi and also amongst leprosy cases. The result of this study is shown below in Table 2.

Table 2

Incidence of HBAg in leprosy, Lepromatous Leprosy and in Healthy army and Civil population.

Categories of	Total Number	HBAg positive sera Total Number	Percentage
Army	929	24	2.6
Civilian Healthy Donors	966	13	1.35
Leprosy all types	152	5	3.29
Lepromatous Leprosy	67	4	5.97

Above table shows that there is no statistical difference in the incidence of HBAg in the Army Personnel and Civil donors, but there is a significant difference ($P<0.05$) between the Army personnel and lepromatous leprosy cases. Similarly, civilian healthy donors show a statistically significant difference ($P\leqslant0.001$) with lepromatous leprosy cases. This high incidence of HBAg in lepromatous leprosy cases has been ascribed to a depression of cell mediated immunity (Dutta and Saha, 1973).

Certain diseases like lymphocytic leukaemia, and Down's syndrome (Blumberg et al, 1971) which show a depressed immune response also have a high incidence of HBAg. But,the high incidence of HBAg in healthy soldiers who have no evident pathology nor any abnormality in their liver functions could hardly be ascribed to a depression of cell mediated immunity. Genetic hypothesis postulated by Blumberg (1972) which states that individuals homozygous for Au gene are susceptible to persistent infection to Australia antigen whilst heterozygous genotypes would develop transient infections when exposed, perhaps may help to answer this riddle of persistent antigenemia in perfectly healthy soldiers. Further, such susceptible individuals who are continuously being exposed to the risk of exposure to HBAg in the service life and barracks may have led to this high incidence rate of HBAg in the trained soldiers.

CONCLUSION

When the incidence of HBAg in trained soldiers with more than 5 years of service was compared with the recruits, it is found that the trained soldiers have a statistically significant higher incidence of HBAg. It is suggested that this may be due to regular periodic innoculation and vaccination that these soldiers have to undergo as a part of their service rules and also to community living with greater chances of exposure. Certain diseases with depressed cell mediated immunity have also a high incidence of HBAg. But, these soldiers do not have such depression of their immune mechanism. Therefore, a genetic predisposition in homozygous people is also suggested as another possible reason for this high carrier rate in the trained soldiers.

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COLD INJURY-AN EPIDEMIOLOGICAL STUDY DURING THE RECENT INDO-PAK WAR

BY

Lt Col P.S. Gill*

The military significance of frost bite is very considerable. In the first world war many thousand cases occured. During the second world war the drastic effects on German forces in Ardennes campaign in 1944–45 are well known. In Korea in winter of 1950–51, 20% of all casualties in the American forces were due to cold injury mainly frost bite. So far as could be judged the casualties among the Chinese and North Korean troops were considerably greater and in certain units there appeared to be 100% incidence of cold injury (Gill 62).

During the last Indo Pak war (Dec 71), in the whole theatre north of Banihal pass the casualties caused by enemy were only a fraction of those inflicted by cold. It is a well known fact of military medicine that in most campaigns up to 1951 non-enemy action casualties have been many times more than those inflicted by the enemy but one could not imagine its repetition in modern times. It is indeed hard to see tough troops, sound of wind and limb, land up in already cluttered up medical units in gloom and despondancy without even a chance to face or fire a bullet in action. These patients have to remain in hospitals for many months leading to a great wastage of man power. It must also be demoralising to a Bn Comdr who along with nearly 40% of his men is struck off in action due to cold injury alone.

Our knowledge of cold injury is still incomplete and there have been no dramatic developments in the treatment. The emphasis therefore should be on its prevention.

All cases brought to a Base Hospital were investigated in detail by a Field Health Org. They numbered 255 and represent nearly 1/3rd of the total cold injury cases which occured in Srinagar-Ladakh Sector. The remaining cases were evacuated through different Base Hospitals. The epidemiological analysis is presented not to find faults but to learn lessons for future prevention. Almost all the cases came from Kargil sector where they were operating at heights varying from 10000 to 12000 ft. The night temp was well below the freezing point and it was very windy on those barren and bleak heights.

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*Then O.C. a Fd Health Org, at present DADH an Inf Div.

Table 1 shows the rank wise distribution of the cases in the present series.

Table I

Rank	No
Offr	—
JCOs	2
Hav	15
Nks	16
ORs	217
NCsE	5
Total	255

This clearly shows that the most sensitive segment to be tackled are the ORs. Is it that the Offrs and JCOs are better clothed and equipped and are more health conscious? Whatever the answer, the fact emerges that more emphasis should be laid on the health education and proper equipment of the ORs. Every regtl offr must ensure that his troops are well equipped for the task to be undertaken. The low incidence among NCsE may be due to lesser exposure inherent in the nature of their duties.

Table II shows the incidence as related to the colour of skin

Table II

Colour of skin	No of cases
V Fair	20
Fair	4
Brown	11
Dark	24
V Dark	196

Though no valid statistical conclusions can be drawn without knowing the colour distribution of the whole population at risk yet it appears that very dark persons seem to be definitely more prone because majority of the troops involved belonged to the middle range viz brown and dark.

Table III shows the occurance of cases in relation to the period of exposure

Table III

Total period of exposure in hours	No of cases	Percentage
24	13	5.1
48	37	14.5
72	60	23.5
more than 72	145	56.0
Total	255	100

Most of the cases occured after prolonged exposure. Besides the human and discipline factor the most important cause appeared to be the breakdown of eqpt like boots due to prolonged strenuous wear and tear. The need for improved equipment and clothing was definitely felt.

Table IV shows the number who did not walk in snow and others who walked on snow

Table IV

		Percentage
No walked on snow	135	52.9
No did not walk on snow	120	47.1
Total	255	100

Table IV(a) shows a further analysis of 135 cases who walked on snow in relation to the no of hrs walked by them before they were afflicted.

Table IV(a)

Period in hrs walked on snow	No of cases	Percentage
8	8	5.2
16	18	13.3
24	30	22.2
more than 24	79	59.3
Total	135	100

It appears that walking on snow per-se was not responsible for frost bite. Out of 255; 120 or 47.1% did not walk on snow and of the 135 who walked on snow 109 or 81.5% suffered only after walking for more than 16 hours. This confirms the conclusions of table III supra.

Table V shows the time lag between noticing the symptoms and the availability of medical aid

Table V

Gap in hrs where medical aid became available	Nos	Cumulative Total	Cumulative Percentage
4	61	61	24.0
8	13	74	29.1
12	28	102	40.2
16	7	129	50.1
20	2	131	50.2
24	66	197	77.6
more than 24	77	254	100

(One patient could not give any definite answer)

It is evident from this study that most of the cases had to wait for many hours to get medical aid. In a difficult terrain and hostile climate the normal 3–4 hourly stage does not hold good and medical aid may not be available for many hours. In this study even after a lapse of 16 hours only 129 or 50.1% had received medical aid and the rest half were still expecting it. The emphasis should therefore be developed on first aid and self help by fellow soldiers, more so because during battles the med centres may be busy and cluttered up with battle casualties requiring immediate and urgent attention. More over tough terrain, the lack of cummunication and Op commitments are bound to cause time lag in evac of casualties.

Every one questioned had at least one item of clothing/eqpt deficient. 78 were deficient of gloves, 74 of scarves and 29 of woolen shirts. In 108 the socks got wet with snow and sweat and they could not change them due to the non-availability of the spares. It is difficult to blame the lack of these items unless one knows the deficiency of clothing amongst those who were also exposed but did not suffer. However the imperative need to fully-equip troops especially in the hostile clime and terrain is quite evident.

A study of the availability of rations showed that out of 255, 148 or 58% were having full rations and did not suffer any shortage, 66 or nearly 26% could have only reduced rations and 41 or 16% had no rations. In a well fed soldier with ample bodily reserves, reduced or temporary lack of rations did not seem to enhance the proneness to cold injury.

Table VI to VIII shows the parts affected and the extent to which they were affected :-

Table – VI

Part affected	Rt Foot	Lt Foot	Both Feet	Rt Hand	Lt Hand	Both Hands
No	17	13	216	6	7	27

TABLE – VII

Affected Toes	1st	II	III	IV	V	ALL
No	113	58	27	21	26	181

Table – VIII

Part	Distal phalanx	Distal and middle phalanges	Whole toe/finger
Foot	61	34	150
Hand	25	4	15

The commonest part affected was the great toe. Unexpectedly there was no difference between the incidence of rt and left hands. Majority suffered the involvement of all three phalanges of the toes/fingers.

These cases were studied for different degrees of frost bite. Out of them 8 or 3.1 % were 1st degree, 204 or 80 % were 2nd degree and 43 or 16.9% were 3rd degree. None was in 4th degree. In this study therefore second degree frost bite appeared to be the commonest cold injury.

Out of 255, 228 or 89.4% had undergone acclimatization. These troops were not rushed in emergency but belonged to one Battalion which had stayed and seasoned for over eighteen months in this area. Acclimatization and seasoning is no substitute for constant vigilance and active prevention. Almost every one said that some lectures were given on acclimatization and prevention of cold injury but no one knew even the elementary basics of prevention. Our present system of health education as a one way lecture seemed to be a complete failure and needs drastic reorientation. The ORs must be explained in their own language and demonstration should be held to ensure that they have understood the basics. Easily understood charts should be supplied centrally by the ACHEB. For instance in US Army to train men in proper use of cold weather clothing many cardinal points have been illustrated by the word "cold" itself and they are.

C Keep clothing *clean* so that it does not *cling* mat down, become greasy and lose insulation,
O *Open* the clothing before you become over heated
L wear clothes *loosely* and in *layers*
D keep clothing *dry*

Training films in prevention and first aid should be made and shown to the troops.

SUMMARY

An epidemiological study of cold injury cases is presented. ORs form the most sensitive segment. Previous service in cold areas or routine acclimatization had no bearing on the incidence of cold injury. Active prevention and constant vigilance are the only remedies. Complete reorientation of our present system of health education with greater and concerted efforts is essential. Before cold induction all deficiencies in eqpt should be made up and cold indoctrination and trg in 1st aid and prevention imparted. Research to improve the eqpt especially foot wear is necessary. Like BPET a cold stress test by prolonged marching in cold/snow should be introduced to find the stress drop out rate and test the efficacy of trg in preventive measures.

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ASEPTIC BONE NECROSIS IN NAVAL DIVERS. PRELIMINARY SURVEY REPORT

BY

Surg LCdr AK Chatterjee*

INTRODUCTION

Decompression sickness is a known occupational hazard of people working in changing ambient pressure — divers, caisson workers, fliers etc. Usual acute manifestations of decompression sickness on or immediately after ascent are well recognised and are successfully treated with therapeutic recompression in most cases. Central nervous system and skeletal systems are, however, susceptible to 'silent' damage by 'silent' bubbles which may not be apparent until months and years later. These lesions though less obvious initially are no less disabing in their ultimate outcome.

Aseptic bone necrosis in compressed air workers was long known as a definite clinico-pathological entity. Painful and disabling juxta-articular lesions affecting joints focussed attention to many cases while others came to light after radiological surveys of asymptomatic groups of compressed air workers (McCallum et al 1966).

Until recently divers were considered to be relatively immune to aseptic bone necrosis. Long exposures to high pressure in compressed air work rather than the short bounce dives were reported by several authors (Grutzmacher, 1941). Sartor (1947); Dale (1952); Ronald (1953); Angei and Cossu (1954); Pirestu and Perra (1960); Barnes (1965); Uhl (1968)).

In recent years a number of radiological surveys of bones of large groups of asymptomatic divers were carried out by several workers in Germany, Japan, Sweden, France, Russia and England. (Herget 1948, 1952); Alnor (1963); Ohta reported by Elliot et al (1969); Slordahl (1953); Fournier et al (1965); Kiryakov (1964); Elliot et al (1969).) A large number of cases of skeletal lesions attributable to high pressure exposures have been detected in these series. The incidence is as high as 23 to 65%. In the age group over 40 years the incidence is 76% in some studies.

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In this context it is well to remember that diving activities have undergone phenomenal changes of late. Deep diving and saturation diving entails very long hyperbaric exposures. Air diving is being utilised more and more increasing the number of dives and total duration of dives per diver.

The concept of pathogenesis in decompression sickness is also acquiring emphasis in new directions in the light of current investigations. Bubbles are considered no doubt to initiate the process but the role of secondary factors such as lipid emboli, clumping and sludging of RBCS etc., have acquired considerable significance in determining the final outcome which includes occurrence of bone necrosis.

The survey reports of bone necrosis in divers quoted above exclude helium dives. In air-helium or oxy-helium dives the risk of decompression sickness—acute or insiduous, are many times more. It is now apparent that the risk of aseptic bone necrosis in divers is real and considerable, though the characteristics of dive which predispose to it are yet to be determined and defined.

MATERIAL AND METHOD

Radiological survey of large joints of both upper and lower limbs of 9 divers of a Clearance Diving Team in a Naval Command were undertaken in 1971. Their diving career extended from 2 to 10 years.

AP and lateral views of 6 joints i.e., hip, knee, ankle, shoulder, elbow and wrist of both sides were taken and scrutinised for the presence of following types of lesions as described by Elliot et al (1969) :—

Dense areas.
Linear or spherical opacities.
Structural failures
Irregular calcified areas.
Translucent areas and cysts.
Cortical thickening.
Degenerative arthritis.

RESULT

No bony lesions were observed in any of the divers.

DISCUSSION

Compared to the high incidences of bony lesions in other series, Elliot et al (1969) in their comprehensive-survey of 350 Royal Naval divers reported only a less than 5% incidence. Other studies recorded as high as 65% incidence.

Absence of bony lesions in Indian Naval divers in the present series was not altogether unexpected because of the following reasons :—

(a) Scrupulous adherence to British diving practice (BR 155) with a proved low incidence of bone necrosis.

(b) Usual avoidance of practices such as diving below limiting line and repetitive dives which increases the risk of decompression sickness.

(c) Diving mostly with oxygen having no risk of decompression sickness or oxygen rich mixtures with lesser risks.

(d) Usual practice of diving for short durations at relatively shallow depths.

In short, their dives are comparatively less complicated in respect of gas mixtures, depth, duration and repetation and carry less hazard of bone necrosis.

However, with the recent expansion of diving activities in the Indian Navy periodical checks of divers will be necessary to confirm the safety of the diving practice not only in respect of acute hazards but also of the insidious lesions which may take time to manifest.

Another study of possible bone lesions in deep divers is currently at hand.

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DEFENCE INSTITUTE OF PHYSIOLOGY & ALLIED SCIENCES AND ITS RESEARCH ACTIVITIES

BY

Surg Cmde MS Malhotra, IN

INTRODUCTION

Research in Physiology as applied to the Armed Forces was initiated in 1950 by establishing a small Physiology Gruop in the Defence Science Laboratory, Research and Development Organisation, Ministry of Defence, New Delhi. The primary objective of this Group was to study by physiological and psychological methods, the best means of increasing the fighting efficiency, safety and comfort of the sailors, soldiers and airmen under varying environmental and operational conditions. The activity also included studies related to different types of rations for troops for general and specialised duties, various types of clothing for tropical and arctic conditions ; to the standards of hearing and vision and other qualities required for particular tasks and to the designing of weapons, equipments and ships with due regard to human capabilities and limitations. As the scope of the work gradually increased, from a small beginning, the Physiology Group was expanded into two Divisions in 1959. During 1960, physiological research cells were also established in the Defence Laboratories at Jodhpur, Landour and Leh to supplement the work of these two Divisions. To cope up with further sharp increase in the demand for physiological research, late in 1962, it was decided to set up a full-fledged institute for physiological research and it was named as "Defence Institute of Physiology and Allied Sciences (DIPAS)".

The Institute was initially located at Madras Medical College, Madras, but due to many technical and administrative difficulties experienced in conducting research from Madras it was shifted to Delhi in December 1968 and is now temporarily located in the precincts of Base Hospital, Delhi Cantt. The site for its permanent location in Delhi Cantt is being examined and there is a proposal to locate it as an independent being in the new Army Hospital complex. In June 1973, the technical and administrative control of the Institute was transferred from the Research and Development Organisation, Ministry of Defence, to the DG AFMS and at present the Institute is under his complete technical and administrative control.

ORGANISATION

The medical services in our Armed Forces being integrate, the Institute is required to work on personnel research problems pertaining to all the three Services. To carry out these duties efficiently and expeditiously, the Institute has been organised into the following Scientific Divisions :—

(a) **Environmental Physiology Division**–consisting of

- (i) Heat Physiology Group
- (ii) Cold Physiology Group
- (iii) High Altitude Group
- (iv) Underwater Physiology Group
- (v) Psychophysiology Group

(b) **Work and Cardiorespiratory Division**–consisting of

- (i) Cardiovascular Group
- (ii) Haematology Group
- (iii) Respiratory Group
- (iv) Work Physiology Group
- (v) Anthropology Group

(c) **Biophysics Division**–consisting of

- (i) Biometeorology Group
- (ii) Neurophysiology Group
- (iii) Biophysics, Sonics & Hearing Group.
- (iv) Clothing Group
- (v) Radioisotope Group
- (vi) Statistics Group

(d) **Biochemistry Division**–consisting of

- (i) General Biochemistry Group
- (ii) Nutrition and Metabolism Group
- (iii) Endocrinology Group
- (iv) Enzymology Group

Each Division is headed by an experienced and highly qualified scientist in the rank of Principal Scientific Officer and each Group is under the charge of a well-qualified scientist in the rank of Senior Scientific Officer Gde I/II who is assisted by Senior/Junior Scientific officer and 4 or 5 scientific assistants. The Institute has thus an authorised establishment of 32 scientific officers and 83 scientific assistants, in addition to adequate administrative and stores personnel. The scientific officers are all experienced scientists with postgraduate degrees like M.D., D.Phil, Ph.D. in different disciplines of science like medicine, physiology, biochemistry, biophysics, electronics etc. Most of the scientific assistants are holders of M. Sc. degree. Quite a few of the officers have been abroad to foreign universities for varying periods.

The research projects allotted to this Institute are all approved by the Armed Forces Medical Research Committee (AFMRC) which functions under the chairmanship of the DG AFMS and has wide membership consisting of eminent civilian scientists and specialists from the Armed Forces Medical Services. The problems for research are either referred to by the users or proposed by the scientists themselves. These are discussed in technical and other details by various Expert Committees of the AFMRC before being finally approved by the main Committee. All the projects accepted by this Committee are, therefore, user-oriented. Besides, there is also considerable scope for fundamental or basic research.

FACILITIES AVAILABLE

The Institute, over the past few years, has acquired a number of modern instruments and equipments required for field and laboratory work on applied physiology, anthropology and biochemistry. Most of the Groups are well equipped and are being further supplemented with the latest instruments for research in their respective fields. The Environmental Physiology Division has two separate climatic chambers – one for simulating hot dry and humid conditions and the other cold conditions for human studies. Besides, there is a temperature-controlled bed where studies on controlled hyperthermia and hypothermia can be undertaken. A decompression chamber with temperature control is available for human experiments under simulated altitude conditions. A recompression chamber for simulating underwater conditions is contemplated. For field experiments, quite a few portable and transistorised instruments are available.

The Work and Cardiorespiratory Division has modern high-speed human treadmill and magnetic brake bicycle ergo-meters with automatic heart rate controller, automatic oxygen and carbon dioxide analysers, and is capable of undertaking advanced research in the field of exercise physiology. The Respiratory Group is equipped with all the modern instruments for conducting detailed lung function studies including blood pH, PO_2, PCO_2 and total body haemoglobin. Equipment for measuring diffusion capacity of lungs has been ordered. The Anthropology Group has got the human densitometer tank, latest measuring calipers and other equipment for studying body composition in man. Facilities also exist for studying the functions of the active sweat glands by plastic impression technique.

The Biophysics Division has facilities for detailed studies on sonics and hearing and measurement of noise and its spectrum analysis. A noise–proof room is being set up shortly for conducting advanced research in sonics and hearing. Multi-channel recorders with different transducers, EEG, EMG and CFF are also available. In addition, a tracer laboratory has been set up to use the radiosotope techniques in physiological and biochemical investigations. For detailed statistical analysis of the data and for proper design of the experiments, mathematicians and statisticians form a Group in the Institute where various electric and electronic calculators are available.

The Biochemistry Division is very well equipped with specialised equipment like spectrophotometer with fluorimeter, ultracentrifuge, high–speed refrigerated centrifuge, thin-layer and gas chromatograph, Warburg apparatus, etc. for any advanced biochemical studies.

The Institute also has a well–equipped workshop capable of repairing, modifying and fabricating new electrical and electronic equipment needed for physiological research. In addition, there is a well–equipped carpentry shop for woodwork. The animal house also forms a part of the Institute for conducting animal experiments.

Views of some of the laboratories using various instruments/equipments are given in Figs. 1 to 6.

RESEARCH ACTIVTIES

A large number of our troops are stationed on our northern borders which extend from the north–eastern borders in NEFA across the entire length of the Himalayas to the north–western borders of Rajasthan. The troops living in these areas are exposed to extremes of climates from sub–zero temperatures to burning hot desert conditions and to varying heights from sea level to altitudes of 20,000 ft. Heterogeneity of population, terrain and the climates in different parts of country from where the soldiers are recruited, pose other problems. Accordingly, most of the work done by this Institute has been in relation to environmental physiology with respect to the effects of heat, cold and high altitude on human body. In addition, studies have been conducted on nutritional problems of troops, load carriage by infantry soldiers, noise and its effects and many other aspects of personnel resarch. Prior to the Chinese aggression in 1962, the main emphasis of work was on the effects of hot environments but after 1962, problems of high altitude and cold form the major part of the work.

Studies on Heat

One of the important studies on heat pertain to the salt requirement in summer. Contray to popular belief, it has been shown by extensive laboratory and field trials that to prevent heat casualties, extra salt in the form of salinised drinks is not required for acclimatized Indians even under the most severe heat and physical activity conditions, as the salt content in their diet is quite adequate for this purpose. What is required is to periodically replace the fluid losses in sweat. This finding has since been confirmed by many workers in other countries. It has been found that a soldier engaged in strenuous tasks under outdoor conditions in summer needs to drink about 1 litre of water of water per hour and, when engaged in moderate activities, only 0.5 litre per hour.

Other studies in this field have been on comfort zone for Indians. The upper level of comfort for them in summer has been found to be 1°F higher than in the case of Europeans. For assessment of heat load on the outdoor workers, a new index called the "Modified Effective Temperature Scale" has been developed which combines air temperature, air movement, humidity, solar radiation and colour of clothing into a single index of warmth. The existing indices do not include the colour of the clothing into consideration. Several studies have also been carried out to determine the effect of high environmental temperatures as encountered in ships, aircrafts and other military vehicles, on the physical and mental efficiencies of the men working there.

The nature of heat casualties occurring in the Indian armed forces has been studied and recommendations on various measures required to control them have been made. It has been observed that most of the heat casualties in the armed forces occur in the Training Centres amongst the recruits. The incidence has been considerably reduced by changing the training schedule so that strenuous outdoor exercises are given during the cool morning and evening hours and indoor training during hot hours of the day. All outdoor work is stopped when the outside temperature exceeds 93°F WBGT.

Extensive studies have been conducted on the habitability survey of various ships in the Indian Navy, most of which have been built primarily for winter conditions but are required to operate in tropical waters. Many structural alterations have been suggested for each class of ship to keep their thermal environments comfortable. Similar studies have also been extended to armoured vehicles and tanks.

A new method of bioclimatic classification of India based on comfort considerations has been evolved and the various military stations requiring air-conditioning have been suggested. This is the first time when such a bioclimatic classification of the country has been done. Controlling humidity in various ammunition workshop depots near the sea coast has been posing serious problems, especially during monsoons. For this a window-type dehumidifier has been designed and fabricated in the Institute. This has been accepted by the Navy for implementation. Studies on ultraviolet component of solar radiation and ozone concentration in relation to habitability and incidence of diseases in high-altiude areas are under progress. Studies are also in progress to determine the ethnic differences in physiological responses to the hot environmental conditions and the maximum tolerance limit of Indian soldirs to various combinations of high temperature, humidity and work. The capacity for continuous and intermittent work under various hot environmental conditions is also being currently evaluated.

Studies at High Altitude

Studies have been conducted on the respiratory and cardiovascular responses of soldiers on acute and chronic exposure to high altitude. The mechanisms involved in high altitude acclimatization and the role of various drugs and chemicals in hastening the acclimatization have been studied. None of the drugs tried like Kcl, iron and other haematinics have been found suitable for this purpose. As at high altitude a soldier is exposed to two stresses of cold and hypoxia simultaneously, it was believed that the soldiers should be acclimatized to cold after their acclimatization to hypoxia had been completed. Our studies have shown that when hypoxia and cold stresses are given together, the acclimatization to altitude is hastened rather than delayed.

Extensive studies bave been done at various altitudes on changes in physical work capacity of a soldier. A reduction of 15% has been seen at 3500 m and 25% at 4000 m, after full acclimatization to altitude has been achieved. The effects of prolonged stay at high altitude on physical work capacity and other physiological parameters have been extensively studied. It has been seen that work capacity drops by about 25% during the first week of arrival at altitude of 3500 m, but slowly improves to reach 85% of sea-level performance by about one year, by which time the acclimatization is fully achieved. The prolonged stay of two years at 4000 m with two months annual leave in the plains, has not been found to cause any ill-effect on cardiovascular, respiratory, liver or other functions. All the changes observed in various systems were adaptive in nature.

Hormonal changes in normals and high altitude pulmonary edema cases at high altitude have been studied. It has been found that in normals the plasma cortisol rises 3-4 folds on the first. day of induction to high altitude and thereafter its level drops slowly to return to almost sea-level values within two weeks. In HAPE cases the initial rise in cortisol level is as in normals, but it drops suddenly to a very low value a day before the occurrence of the disease.

Changes in metabolism of carbohydrates, proteins and fats have also been studied. A rise in fasting blood sugar level has been found to occur during the firsn 10 months of stay, but it returns to original sea-level values after that period. No change in fat

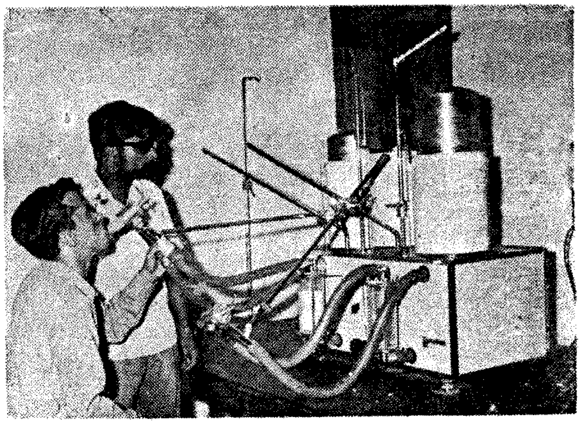

Fig. 1 Lung functions being measured in Pulmotest

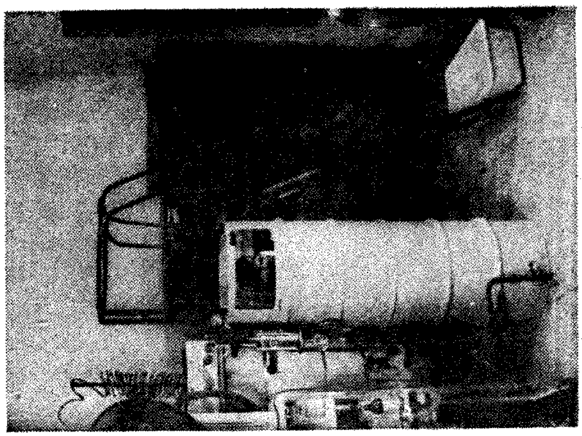

Fig. 2 Human densitometry tank

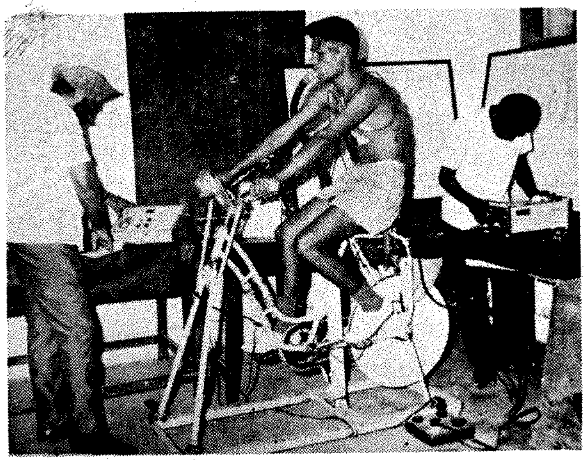

Fig. 3 Bicycle ergometer is being used for measuring physical work capacity

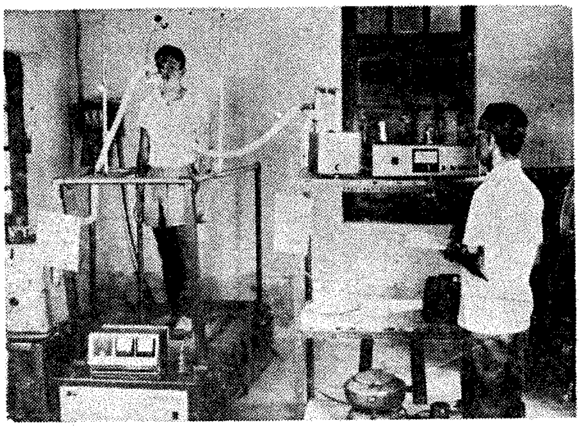

Fig. 4 Ventilation and oxygen consumption being measured during controlled exercise on treadmill

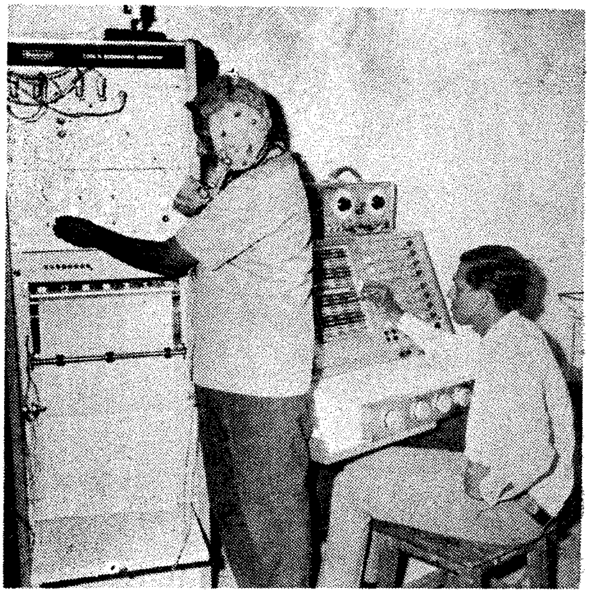

Fig. 5 Polygraph and Electroencephalogroph in use in the Neurophysiology laboratory

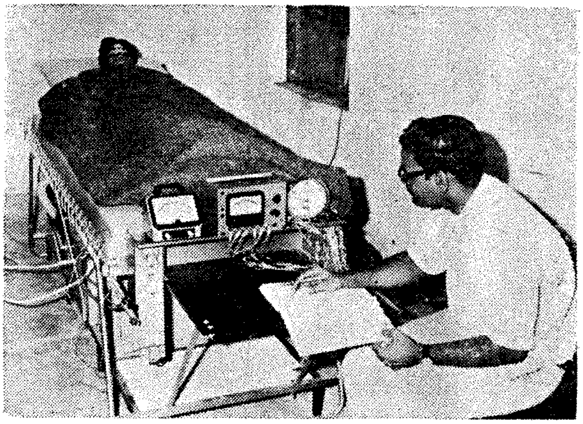

Fig. 6 Physiological responses being tested during hyperthermia in a temperature-controlled bed

metabolism has been seen, but the total plasma proteins have been found to decline. There is also reversal of albumin–globulin ratio with serum albumin showing a decline and globulin a rise.

Studies on Cold

Our studies on cold acclimatization have shown that shivering response of newcomers to cold environments is more pronounced at high altitude that at sea level, when exposed to identical temperature conditions. Further, it has been found that the natives of high altitude maintain their body temperature in cold by non-shivering thermogenesis unlike the newcomers who do so by shivering. Extensive studies have been done on prediction of susceptibity to frostbite and it has been found that soldiers who have a high heat output from hands on immersion in cold water, are less prone to cold injury. It has been further seen that general cold acclimatization helps to reduce the risk of frostbite. Currently, experimental frostbite is being produced in animals to determine the effect of various factors on its genesis.

Studies in Work Physiology

One of the important studies in the field of work physiology has been on the problem of load carriage by an infantry soldier. The maximum load that can be carried by a soldier without seriously affecting his performance and causing undue fatigue, depends upon a number of factors, such as, the speed of marching, the terrain, the climate, the gradient, the altitude, etc. Of these, the optimum speed of marching and the proper distribution of the load on the body are the two most important factors in reducing fatigue. Another related study has been on the best manner of carrying school bags by children. Of the various methods of carriage, the rucksack method has been found to be the most economical and consequently the best, and the hand carriage, which requires about 250% more energy than the rucksack method, the worst.

A new point system of physical training has been suggested to be introduced in the Indian army for officers working in sedentary jobs and in places where there are no facilities or suitable grounds and equipment for imparting organised physical training. The proposed technique includes both the isometric and isotonic free–hand exercises. Extensive studies have also been done on the physiological assessment of the top Indian athletes and sportsmen. Various physiological functions connected with athletic ability have been measured. These studies have also helped in identifying the deficiencies in various funtions in the players and provide guide for their training in proper direction.

Ageing Process in Indians

It has been found that ageing process in Indians, as evidenced from capacity for maximum work, muscle power, aerobic and anaerobic capacity, etc., starts to manifest at an early age of 30 years. There is a further noticeable-fall in physical work capacity at the age of 37 years. Various mental facuties, on the other hand, have been found to improve till 35 years of age and this level is maintained thereafter till the age of 45 years up to which these studies have been conducted. Based on these findings, standards for battle physical efficiency tests have been revised and norms have been laid down for different age groups, separately for fighting arms and supporting services.

Food and Nutrition Studies

Extensive studies have been carried out to determine the energy cost of various activities under different climatic conditions in the plains as well as at high altitude. During the course of these studies, two very significant facts have been revealed; firstly, it has been found that the energy cost increases proportionately with body weight, and secondly, that the environmental temperature has little effect on basal and work metabolism. Studies have been carried out to determine the calorie and food requirements of troops at moderte and high altitudes. Investigations have also been carried out on the requirements of food under special service conditions, such as, survival rations for shipwrecked sailors, hard scale rations for ski troops, rations for divers and submariners, etc. Rationalisation of existing army ration scales both for field and peace areas have also been done. Further work is in progress to assess the nutritional requirements of recruits during their training period.

Eye Movement studies

Studies have been completed to determine the degree of deterioration in visibility when there is a relative motion between the observer and the objects. Such a situation arises when a high-speed aircraft is flown at low altitudes. Studies of eye movements in response to moving objects and on the speed of accommodation were also conducted in order to devise suitable objective tests to indicate the changes in states of consciousness of individuals exposed to single or a combination of stresses.

Noise and Sonic Studies

Extensive studies have been done on noise intensities to which service personnel are exposed and the effect of this noise on their hearing acuity in the Navy as well as in the Army. The noise problem has been found to be of greater magnitude on board INS Vikrant where the aircrafts are required to take off from the flight deck within a distance of 30 yds. The noise level up to 120 dB affects not only the sailors working on the flight deck but a deck below also. Noise of the engine room in various ships has been also found to be much above the safe limit and is very damaging. In the army, the problem has been found to be acute in gunners who have to fire guns which produce high-intensity noise as blast. Hearing of a large percentage of the personnel exposed to these environments has been found to be affected. Suitable protective measures have been recommended where needed, and work is in progress to devise suitable ear defenders indigenously.

Anthropometry

The Anthropology Group of this Institute has conducted extensive anthropometric survey of the pilots as well as of other ground duty air force personnel belonging to different ethnic groups in connection with the sizing of personnel equipment and the work space requirments in the cockpit of modern fighter aircraft. Changes in body composition of troops at high altitude have also been studied using anthropometric, roentgenogrammetric and densitometric techniques. Relationship of body composition with physical work capacity is being examined at present.

Psychophysiology Studies

Problems relating to the psychophysiological changes which accrue from various enviroumental stresses operative in military situations have been extensively studied. The effects of high altitude on general efficiency have revealed a decline in learning ability of younger age groups at an altitude of 3500 m, whereas other mental functions like numerical ability, perceptual speed and immediate memory have not shown any deterioration. No incapacitating effects on the overall performance efficiency of jawans have been recorded at an altitude of 4000 m over a stay period of two years. Currently, studies are under way to investigate the effects of heat and cold on certain complex mental functions like associative learning, reasoning ability, discriminative judgement and mental alertness.

From the above, it would be apparent that this Institute is unique in that so many aspects of physiological and biochemical research like that on altitude, heat, cold, physical fitness, nutrition, aviation physiology, underwater physiology, anthropology, psychophysiology, are being done in one laboratory under one roof. Combining all these disciplines into one laboratory ensures intimate and close collaboration and frequent exchange of views between the scientists working on various aspects of the studies which is necessary and vital for research. It is also economical as the equipment, instruments, workshop and other services are not duplicated.

ACKNOWLEDGENENT

The author is thankful to AVM JHF Manekshaw, Director of Medical Research, Office of the DG AFMS, for suggesting the topic.

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PRIMARY HYPERLIPOPROTEINEMIAS WITH A CASE REPORT

BY

Col K.K. Gupta* and Maj Yashpal**

The association between dietary fat, blood lipids and arterial disease still remains complex. For better understanding of the problem, various types of plasma lipids have been identified. The lipids in plasma of clinical importance are Cholesterol, triglycerides and free fatty acids (FFA). All circulate in plasma bound to proteins, the FFA with Albumin, the Cholesterol and triglycerides along with phospholipids as lipoproteins. By the use of ultracentrifuge and the electrophoresis lipoproteins can be separated in four major classes. High density (H D L or Alpha lipoprotein, Sf 0–2), low density (L D L or beta lipoprotein, Sf 0–20) very low density (VLD L or pre beta lipoprotein Sf 20–400) and Chylomicrons (Sf 4000). Most of the triglycerides are carried by Chylomicrons and V L D L and most of the Cholesterol in H D L.

Disturbance of lipid metabolism is better understood when considered in terms of lipoproteins, the form in which nearly all lipids except free fatty acids are present in plasma. Both selective increase and deficiency of some of the groups or classes of lipoproteins can occur. This paper presents a case of hyperlipoproteinemia of type III and a short discussion of diagnosis of various types of hyperlipoproteinemias.

CASE REPORT

A middle aged lady 43 years of age came under observation with the history of occipital headache, palpitation and frequent insomnia of 4 years duration with occasional Anginal pain 2–3 times a month for last 4 months. Family history of Xanthomatosis over the elbows in two younger sisters with hypercholesterolemia was present.

On examination the patient was obese with height of 155 cms and weight 76 kg. Pulse, temperature and respiration were within normal limits. Basal BP was 220/120 mm of Hg (lying). There were no signs of cardiac decompensation. Examination of skin revealed tuberous Xanthomas over the elbows, knees and Xanthelasma on the upper eyelids. Heart sounds were normal all over except for loud A2. No murmur, gallop or triple rhythm was audible. Lungs clinically and radiologically were clear. Fundus showed Grade II hypertensive changes and other systemic examination revealed nothing abnormal.

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Result of urine examination was normal. Blood urea was 36 mgm %. Uric acid 2 mgm% and GTT was within normal limits. Serum cholesterol was found to be 950 mgm% and triglycerides (VDID) 1025 mgm%. Chilled serum appearance after overnight stay in refrigerator was opalescent. Lipoprotein electrophoresis showed a "Broad–Beta" band. Serum transaminase level was within normal limits. ECG was consistent with diagnosis of anterolateral ischaemia. X–Ray chest showed normal cardiothoracic ratio with unfolding of Aorta.

The diagnosis of Primary Hyperlipoproteinemia of Type III was made in view of the following findings :–

(a) Tuberous Xanthoma in a middle aged woman.
(b) Family history of Xanthoma and Hypercholesterolaemia in two sisters.
(c) Evidence of anterolateral ischaemia in heart.
(d) Opalescent serum.
(e) Combined Hypercholesterolemia and Hypertriglyceridemia.
(f) Broad beta band in lipoprotein electrophoresis.

DICUSSION

Fredrickson, Levy and Less (1967) classified Hyperlipoproteinemia in 5 types. World Health Organisation (1970) modified this classification into Primary and Secondary. The Secondary Hyperlipoproteinemia is common, important causes being diabetes mellitus, alcohol, Chronic renal diseases and Hypothyroidism. No lipoprotein pattern is specific for any of these diseases and the pattern my vary from time to time in the same patient (*B M J* 1973).

Primary Hyperlipoproteinemias are of the following 5 types :–

Type I :– It is due to deficiency of the lipoprotein lipase leading to accumulation of Chylomicrons. The disease starts in childhood and is manifested by recurrent attacks of acute pancreatitis (Friedrickson et al 1966). Pre–B–lipoproteins (VLDL) may be raised but there is decrease in A and B lipoproteins (HDL) and LDL). Diagnosis is suspected in a young patient having repeated attacks of severe mid–abdominal pain. Triglyceride concentration is increased far more than that of Cholesterol and Chylomicron band predominates on electrophoresis.

In chilled serum there is a creamy layer on the top and the infranatant serum is limpid. Treatment consists in cutting down the dietary fat, both saturated and unsaturated to 30 grams per day.

Type II :- It is inherited as mendelian dominant trait and is characterised by increased concentration of LDL. Patient usually presents with Xanthomas in the tendons, tuberous Xanthomas on elbows and kness, Xanthelasma and early onset of corneal circus. Chilled plasma is clear and there is hypercholesterolemia of more than 400 mgm % without hypertriglyceridaemia.

Treatment consists in reduction of cholestesterol and saturated fat in diet and use of cholesterol reducing drugs such as cholestyramine 15–30 grams a day or clofibrate one gram a day.

Type III :- The characteristics are well demonstrated by the case described by us. A middle aged patient presented with Xanthoma tuberosum on knees and elbows and Xanthelasma on the eyelids. Family history of similar complaints was present in two sisters. There was evidence of atherosclerosis in the form of anterolateral ischaemia of myocardium. There was combined hypercholesterolaemla and hypertriglyceridaemia. The serum was opalascent and broad beta band was seen on electrophoresis. The treatment in these cases consists in restriction of simple sugars and cholesterol in diet but not of fats which provide 40 to 50 percent of calories. With this treatment serum cholesterol came down to 290 mgm% and Triglyceride to 340 mg% after four months. Size of Xanthomas was much reduced.

Type IV :- It is characterised by accumulation of VLDL of normal composition and electrophoretic mobility. Both A and B lipoproteins are depressed. Most of the patients are obese. They may report with erruptive Xanthomas, recurrent pancreatitis or with premature vascular disease. Fasting serum is opalascent on standing during night in refrigerator. Triglyceride level is increased. Few patients will have hyperglycaemia. The treatment consists in dealing with obesity by cutting down total calories, amount of simple sugars and replacement of saturated fats with unsaturated ones.

Type V :- This is a rare disorder of middle age in which both chylomicrons and pre–B–lipoproteins are raised. The clinical features consist of obesity, insulin independent diabetes mellitus, erruptive Xanthomas and recurrent acute pancreatitis. The incidence of atherosclerosis surprisingly is not high. The serum on standing overnight in refrigerator shows an upper creamy layer due to increased chylomicrons and opalascent infranatant serum due to increased triglycerides.

There may be severe hyper glycaemia with ketoacidosis. Hyper uricaemia is also common.

Treatment consists in cutting down calories intake for obesity and excess of sugars and fats. The hyperglycaemia usually responds to calories restriction and addition of sulphonylurea drugs.

From above description it will be observed that for a complete lipid profile in a patient sophisticated apparatus for ultracentrifuge and immuno-electrophoresis is required. But for clinical diagnosis of various types of hyperlipoproteinemias in an ordinary hospital estimation of serum cholesterol, triglycerides and simple observation of serum standing overnight in a refrigerator may be adequate (Bary Lewis–1973). From therapeutic approach, some physicians are still sceptical about the evaluation of role of blood lipids in arterial diseases. Dennis Krikler (1973) presents a more reasonable approach to the problem by stating that there is such a clear association between hyperlipidaemia and vascular disease that advice on the lowering of blood lipid levels is sensible.

SUMMARY

A case of primary hyperlipoproteinemia type III has been described. Main features of the case were obesity, xanthoma tuberosum, xanthelasma palpaberum, anterolateral ischaemia of heart, hypercholesterolaemia, hypeprtriglyceridaemia and broad B band in electrophoresis. A brief review of various types of primary hyperlipoproteinaemia has been given, describing their clinical features and diagnostic criteria.

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A CASE OF FASCIOLOPSIS BUSKI INFESTATION IN UTTAR PRADESH

BY

Maj S.S. Chandra* Dr. C.M.S. Siddhu**
Dr. S.N. Pandey* and Dr. Pramod Nath******

INTRODUCTION

Infestation with the giant intestinal fluke, Fasciolopsis buski, in man is a medical curiosity and paucity of information available in the literature regarding this parasite is a striking feature. Stoll (1947) has reported that about 10 million people in Asia were suffering from F. buski infestation and almost half of these were from China. However, it has also been found in limited areas of Thailand, Vietnam, Laos and East Pakistan. Investigations were carried out in India by Buckley in 1939 who found 59.7 per cent of the people in Assam were infestated with F. buski. Similar studies were also carried out by Satya Prakash, Ghose and Varmani (1967) in Ghaziabad district of Uttar Pradesh who failed to find a single human infection. Human fasciolopsiais is has never been reported from this province except that of Lal (1923) who reported a single isolated case from a district hospital at Bullandshar. The present communication deals with a case of fasciolopsis buski infestation in a child attending primary health centre at Sarojini Nagar in Uttar Pradesh.

CASE REPORT

R. R., a girl aged 12 years from village Shiv Singh Khera of Unnao district attended Sarojini Nagar Primary Health Centre, Lucknow, with the complaints of griping pain in epigastric and umbilical region. She also complained of loose motions, loss of appetite, distension of abdomen and passing of leaf–like flat worms in the faeces. The symptoms started insiduously about four weeks before and gradually became aggravated. During last three days she became very weak, pale and debilitated. She belonged to a poor family and main items in the diet were chapati and dal. She also gave the history of eating raw lotus tubers and sugarcane stems directly collected from a big perennial lake situated about half a kilometer from her residence.

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On examination, she was found restless, and toxic. She was pale, anaemic and poorly nourished. The pulse was 82 per minute, respiration 20 per minute and body temperature was 98.4°F. The blood pressure was 108/60 mm of Hg. Oedema was present over the ankles. During the systemic examination the abdomen was found markedly distended with tenderness over epigastric and umbilical regions. Liver was palpable 5 cm. below costal margin. The spleen was not palpable. There was no evidence of fluid in abdomen. Other systems were found normal.

During laboratory investigation her haemoglobin concentration was 7.5 gms per 100 ml of blood and total leucocyte count 12,800/cumm. The differential leucocyte count showed polymorphs 75 per cent, lymphocytes 11 percent and eosinophil 14 percent. The erythrocyte sedimentation rate was 20 mm for 1st hour. Routine examination of urine did not reveal any abnormality. Freshly passed samples of faeces demonstrated several leaf-like flat worms measuring 3 to 6 cm. Microscopic examination revealed plenty of eggs of F. buski. The average count by direct smear method was 12 eggs per smear of faeces.

She was given two tablets of Piperazine adipate (0.3 gms each) orally three times daily followed by Mag Sulph purge for the first three days after admission. She passed 6 to 15 flukes in each motion and a total of 301 worms during three days stay in the hospital. She felt great relief of her symptoms on the third day. The dose of piperazine was reduced to one tablet three times daily from the fourth to seventh day when she passed 15 flukes more and finally stopped passing any more. Following this on eighth day, her abdomen became soft with disappearance of distension. Her appetite improved and she became cheerful. She was discharged from the hospital and was called after one week. Her stool was re–examined after a week which revealed ova of F. buski in moderate numbers. She was now given 5 gms of Alcopar orally in empty stomach with one ounce of mag sulph purge when she passed 6 more worms during the next 48 hours. She was given iron and multi–vitamin tablets for one month following which there was an all round improvement in her health and repeated examination of stools also revealed no ova of F. buski. Total number of flukes passed by the girl was 344 in the hospital. During the follow–up medical check up for one year she remained healthy.

MORPHOLOGY OF ADULT WORM AND EGGS

In freshly passed faecal samples, large flat leaf–like pinkish or cream–coloured worms were seen. Most of them were alive showing contractions. Some of the worms were smaller in size but majority of them measured between 35 to 75 mm length and 15

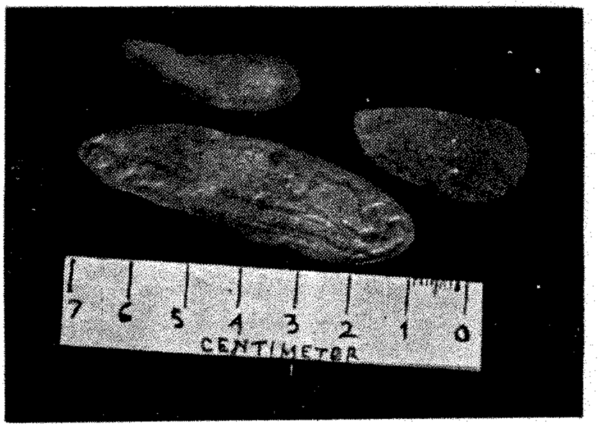

Fig. 1 Photograph of adult Fasciolopis buski from stool of patient showing different shape size and gross appearances

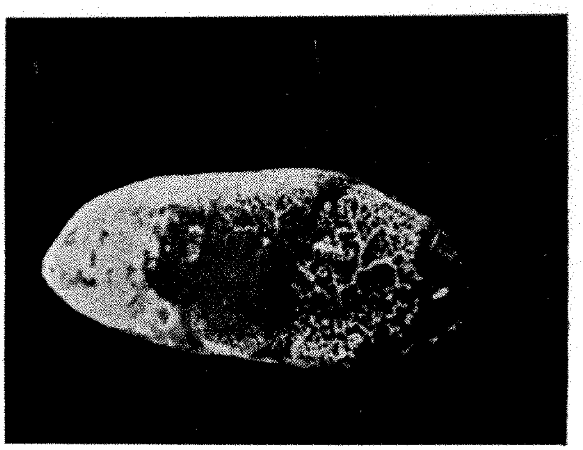

Fig. 2 Photograph of stained adult F. buski showing dendritic testes

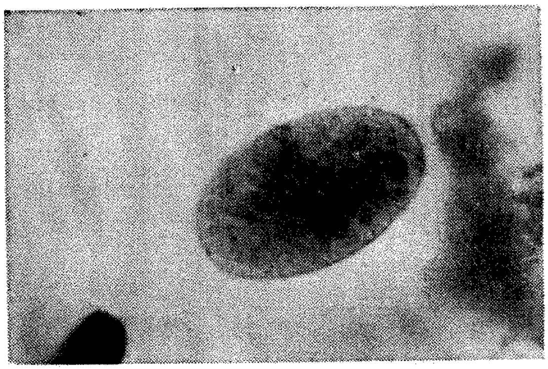

Fig. 3 Microphotograph of stool smear showing ova of F. buski

to 20 mm breadth with a thickness between 2 to 4 mm. The margins were wavy and thin with concavity on the ventral surface. A small oral sucker was situated ventrally at the anterior end while a bigger one, close to the oral sucker (Fig.1). Examination of the worm fixed in formalin in between two glass slides and stained with the acetoalum-carmine demonstrated conspicuous genitalia with dendritic pair of testes occupying almost two-fifth of the posterior end of the worm (Fig. 2). The intestine was prominent with the two unbranched ceca running a wavy manner towards the posterior end along the two lateral margins. The eggs were large, oval in shape and golden brown in colour. They measured from 130μ to 140μ in length and 80μ to 85μ in breadth. The umembryonated eggs had a transparent shell with a small slightly convex operculum at one end. Protoplasm inside the eggs was divided into a large number of regular globular shiny masses which almost filled the entire space in the shell (Fig. 3).

DISCUSSION

The discovery of Fasciolopsis buski in this case was taken to the great surprise because the parasite has never been previously reported in the human beings from this area. Therefore due attention should be paid towards its source of infection and epidemiological significance. Fasciolopsiasis seems to be restricted to areas of world where people raise water plants like water caltrop, water chestnut and lotus in the presence of snails required for parasite to complete its life cycle. The uncooked metacercariae-laden water plants when consumed by the people causes infection in them. Therefore, the infection is found commonly in the individuals and families that live in the immediate vicinity of edible water plants and the prevalence rate is highest amongst the children. The present case also belongs to a poor family with a history of consumption of water chestnut and lotus. The evidence has been found in the child. The source of infection seems to be the lake in the village. The family and population around the lake requires a thorough investigation.

Adult parasites, in man usually reside in the duodenum or jejunum and rarely in the pylorus or colon. They may cause local areas of inflammation and sometimes ulceration and haemorrhage. The clinical signs and symptoms may vary from asymptomatic state in persons harbouring few worms to severe forms showing marked toxicity, diarrhoea, dysentery, abdominal pain, oedema and the passage of undigested food in the faeces. Abdominal pain sometimes mimics peptic ulcer pain. Goddard (1919) has studied these cases and has classified the clinical course in three stages.

I. **Period of latency :-** During this stage the patients remain asymptomatic for many months and subsequently towards the end of this stage may develop mild anaemia, asthenia and gradually passe into second stage.

II. **Period of diarrhoea** :– This is the stage when patient seeks doctor's advice. Initially the patient starts passing loose motions, about 5 to 6 times a day which may continue for months. There may be periods of intermissions in between. Anaemia becomes marked now. Diarrhoic stool is usually pale–yellow, foul smelling and contains lot of undigested food matters. In infants and children, protuberant abdomen may be the first evidence of the disease.

III. **Period of oedema** :– In the later stages, the patient may develop marked anaemia and oedema which first appears in the abdomen and extend to extremities and other parts including face. Skin becomes tough, dry, harsh and pale. The patient becomes extremely weak and gradually dyspnoea supervenes. Ultimately signs of incompetancy of heart appears and patient dies of exhaustion. Cole (1922) stated that the disease may be fatal unless parasites are expelled by art or by accident and that rapid recovery follows when the parasites are evacuated before the latter stages of exhaustion set in.

SUMMARY

The present communication deals with a case of fasciolopsiasis in a young girl, resident of Unnao district, Uttar Pradesh.

ACKNOWLEDGEMENT

We are grateful to Dr. (Mrs.) Premvati, Department of Zoology, Lucknow University for species identification of the adult fluke.

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A STUDY OF HIGH ALTITUDE CEREBRAL OEDEMA
(with case report)

BY

Maj A.I. Lazar*

INTRODUCTION

Thanks to the pioneering work done by various workers all over the world into the pathogenesis, criteria for clinical diagnosis and treatment of high altitude pulmonary oedema in the last few years, its prompt control and cure under effective treatment is mostly a matter of routine now and its once lethal potential has, happily, become a thing of the past. Though cerebral oedema is a recognised and expected complication of high altitude pulmonary oedema – syndrome, it is invariably mild and silent clinically that the vast majority of cases of high altitude pulmonary oedema do not require institution of specific therapy separately against cerebral oedema.

Clinically overt and gross cerebral oedema of high altitude is, however, exceptionally rare compared to the more frequent and evident high altitude pulmonary oedema. Treatment of these rare cases is different from the routine treatment of high altitude pulmonary oedema and will demand therapeutic measures specifically designed to relieve cerebral oedema.

A young Sikh soldier who suffered from overt cerebral oedema of high altitude with coexisting pulmonary oedema and showed complete recovery under treatment provided fine opportunity to probe into its clinical presentation, principles of treatment, probable pathogenesis and reasons for its striking rarity.

CASE REPORT

A 23 year old Sikh soldier, after ten days of casual leave in the plains of Punjab, went straight to his unit in the high altitude area (14000 Ft) on 26th Feb 73, without observing the required acclimatisation schedule. Alas, the price he paid for this seemingly minor lapse was indeed heavy and memorable. He was struck down the very next day, with alarming dyspnoea and rapid deterioration in the level of consciousness.

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He was received in this hospital on 27th Feb 73, (1730 hrs) in a comatose state showing marked cyanosis, tachypnoea and cold extremities with no recordable pulse or B–P. Bilateral, extensive coarse crepitations were heard over both pulmonay bases. X–Ray chest confirmed the clinical diagnosis of high altitude pulmonary oedema (RMZ, RLZ and LMZ). Energetic and prolonged resuscitation with infusion of fluids (via venesection) pressor agents (continuous Mephentine drip) along with IV Lasix (80 mgm) wymesone (4 mgms) O_2 inhalation and antibiotics (Sodium Penicillin) after a lapse of 8 hrs, finally revived perceptible pulse and B–P, though both were still far from satisfactory. Rapid, intravenous digitalisation (Digoxin 1 mgm) was added then and continued at (0.5 mgm) eight hourly intervals. While the above treatment after 24 hrs restored a normal B–P, relief of cyanosis, an appreciable reduction in pulmonary adv. sounds and improvement in the rate/volume of pulse, the patient remained comatose and often showed irritable behaviour in addition to dilated and sluggish pupils. There was hyporeflexia in general. There was no evidence of neck–rigidity or focal neurological deficits. Fundoscopy showed congestion and blurring of optic disc–margins with no exudates or haemorrhages. C.S.F.–Pressure (200 cms of C.S.F.).. NAD–otherwise

The above clinical picture indicated the possibility of coexisting cerebral oedema which is gross and dominant compared to pulmonary oedema. Specific measures, as follows, were soon instituted to combat cerebral oedema, in addition to maintenance doses of lasix, and antibiotics.

(a) Rapid infusions of Mannitol (350cc in ½ hr)
Total Mannitol given – 1050cc in 12 hrs.

(b) More intensive steroid therapy (Dexamethasone) 8 mgms I.V 6 hrly x 3 days and very carefully tappered off gradually later.

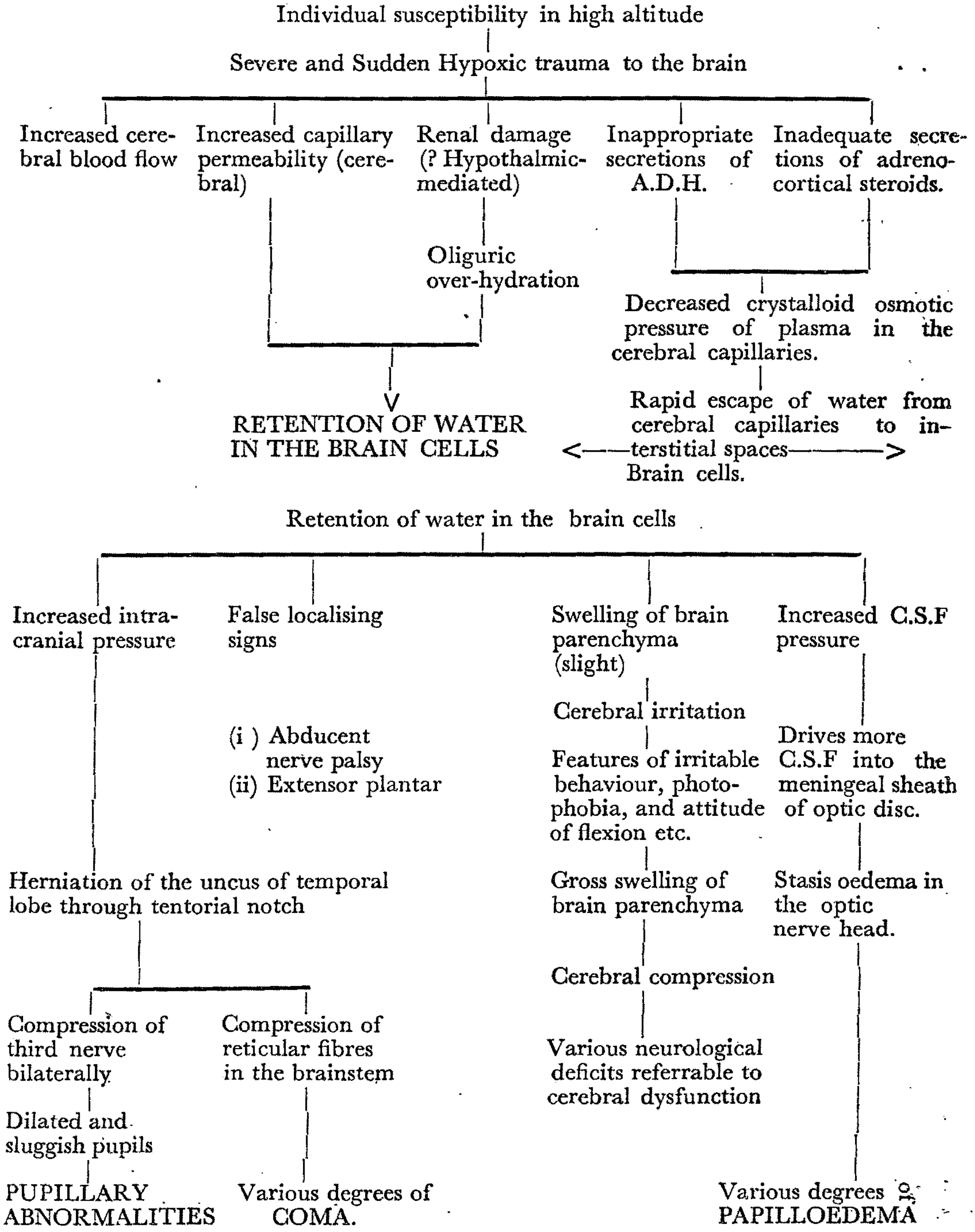
(c) Avoiding hypotonic solutions for transfusions.

(d) Reduction of fluids (Transfusion) to the barest minimum.

Isotonic Saline	540 c.c	in 24 hrs x 3 days.
25% Glucose	540 cc.	

The above schedule continued for the next 72 hrs, finally succeeded in restoring gradually but steadily, a fully conscious patient free from any symptom or abnormality. He was discharged on sick leave. He is serving again in high altitude area after sick leave.

PROBABLE PATHOGENESIS AND MARCH OF EVENTS


Individual susceptibility in high altitude
Severe and Sudden Hypoxic trauma to the brain
Increased cerebral blood flow
Increased capillary permeability (cerebral)
Renal damage (? Hypothalmic-mediated)
Inappropriate secretions of A.D.H.
Inadequate secretions of adreno-cortical steroids.
Oliguric over-hydration
Decreased crystalloid osmotic pressure of plasma in the cerebral capillaries.
Rapid escape of water from cerebral capillaries to interstitial spaces
Brain cells.
RETENTION OF WATER IN THE BRAIN CELLS
Retention of water in the brain cells
Increased intracranial pressure
False localising signs
(i) Abducent nerve palsy
(ii) Extensor plantar
Swelling of brain parenchyma (slight)
Cerebral irritation
Features of irritable behaviour, photophobia, and attitude of flexion etc.
Gross swelling of brain parenchyma
Cerebral compression
Various neurological deficits referrable to cerebral dysfunction
Increased C.S.F pressure
Drives more C.S.F into the meningeal sheath of optic disc.
Stasis oedema in the optic nerve head.
Various degrees of PAPILLOEDEMA
Herniation of the uncus of temporal lobe through tentorial notch
Compression of third nerve bilaterally
Dilated and sluggish pupils
PUPILLARY ABNORMALITIES
Compression of reticular fibres in the brainstem
Various degrees of COMA.

COMMENTS

This case naturally reminds the importance of proper acclimatisation before entering the high altitude area especially after short stay at low altitude. Combination of cerebral oedema and pulmonary oedema made this patient's condition particularly grave and gloomy with persistence of comatose state for about four days.

The striking rarity of overt cerebral oedema of high altitude compared to pulmonary oedema, may be due to the qualitative difference in the permeability–properties of cerebral capillaries. It appears that cerebral capillaries are less susceptible/permeable to the anoxic stimulus compared to pulmonary capillaries and only sudden and prolonged hypoxia/anoxia seems to affect cerebral capillaries.

This is all the more paradoxical and significant because pulmonary capillaries normally have the lowest capillary pressure (8 mms Hg) in the body which acts as a deterrent and natural safe–guard against pulmonary oedema. But, this natural safeguard is altered critically in cases of high altitude pulmonary oedema due to immense increase in pulmonary capillary pressure secondary to selective vasoconstriction of pulmonary venules. The fact that pulmonary alveoli have no lymphatic drainage is an additional handicap and invitation to pulmonary oedema.

The principal internal turnover of fluids in the brain (cerebral capillaries———> Choroid plexus———>C.S.F.———>venous reservoirs) with its wider scope for accommodation and circulation compared to the very limited scope to the pulmonary circulation also explains the relative immunity to cerebral oedema.

Routine treatment against pulmonary oedema completely failed to relieve associated cerebral oedema and a different range of drugs became necessary. Rapid and repeated infusions of Mannitol, restriction of fluids, avoidance of hypotonic solutions for transfusions along with intensive and prolonged steroid therapy (Dexamethasone is the steroid of choice) as outlined above, are the remedial steps to combat cerebral oedema specifically, as illustrated by this case. The cardinal principle and aim in the treatment is to establish an osmotic gradient across the blood–brain barrier that removes water from the brain and reduces cerebral oedema. Impaired permeability of cerebral capillaries is corrected by steroids. However, withdrawal of steroid has to be very gradual and most carefully planned in this case as acute steroid–withdrawal can cause rebound effect of further cerebral oedema immediately and also lead to "Benign intracranial hypertension" or what is being currently designated as "Reducted C.S.F. absorption syndrome" later on by increasing RAV and upsetting the following formula on C.S.F. absorption (IAN Johnston 1973)

$$F_{C.S.F.} \propto \frac{P_{C.S.F.} - P_{S-S}}{R_{AV}}$$

$F_{C.S.F.}$ (Flow of C.S.F. across the arachnoid villi).
$P_{C.S.F.}$ (C.S.F. pressure in the sub-arachnoid space).
P_{S-S} (Sagittal–Sinus pressure)
R_{AV} (Resistance to flow across the arachnoid villi).

Hyper–baric Oxygen therapy can be additionally effective and beneficial in relieving high altitude cerebral oedema, if available.

Rapid digitalisation, while not required in the routine, mild cases of high altitude pulmonary oedema, does confer additional benefits and accentuation in the elimination of severe pulmonary oedema and improvement in the circulatory efficiency. It appears to be resulting from enhanced myocardial contractility leading to increased stroke volume and reduction in pulmonary congestion.

SUMMARY

An unusual case of high altitude cerebral oedema with coexisting pulmonary oedema in a young male is reported. Clinical features, pathogenesis, specific treatment and rarity of high altitude cerebral oedema are briefly discussed.

ACKNOWLEDGEMENT

I am grateful to Col S. Venkataraman, VSM, ADMS, 27 Mtn Div and Lt Col M.L. Pramanik, OC 327 Field Hospital for their kind permission to publish this case. I am also indebted to Capt C.M. Gupta, M.O. for the valuable assistance in the management of this case.

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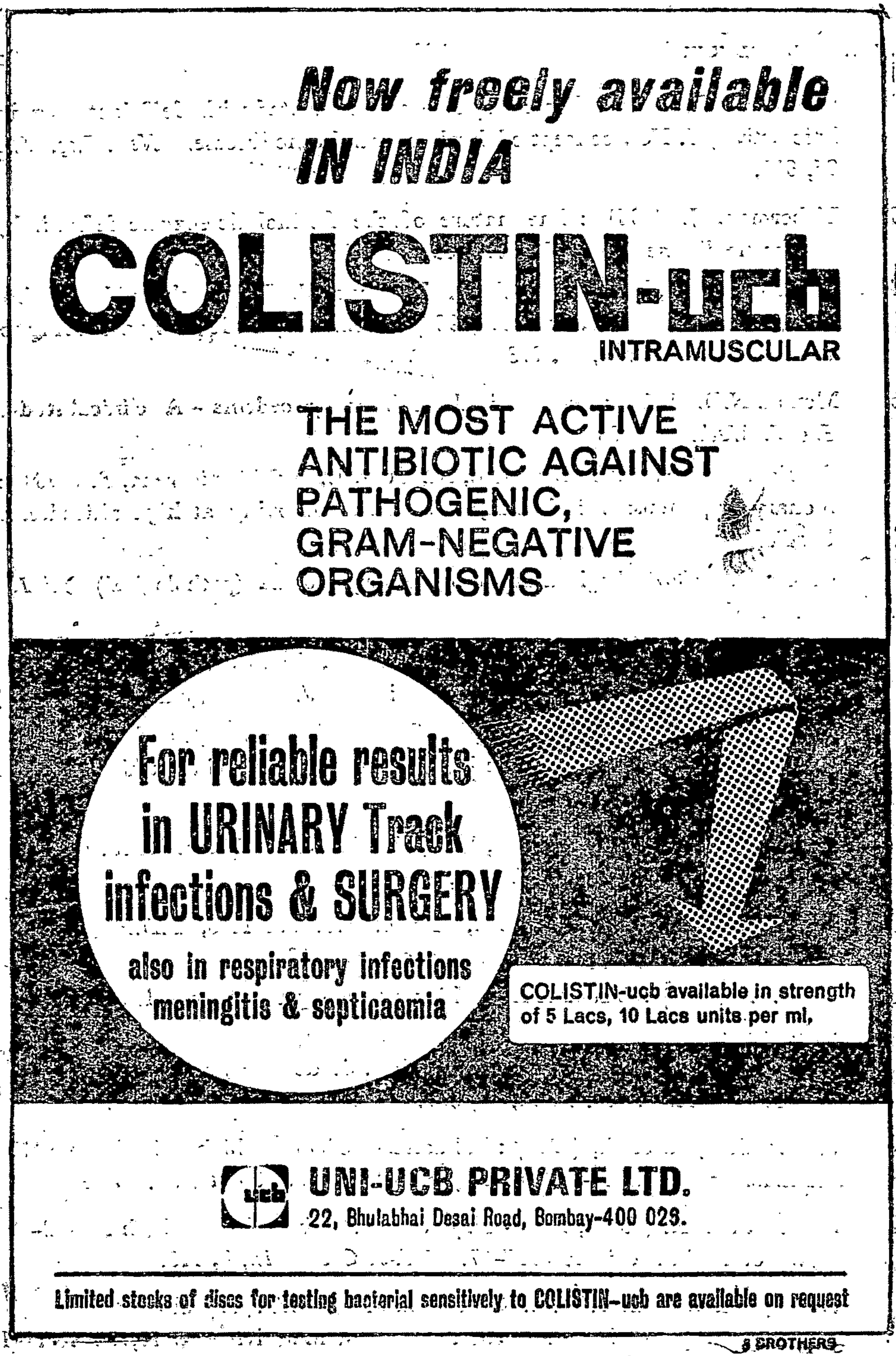

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S BROTHERS

G.S.W. FACE AND TONGUE – AN INTERESTING CASE REPORT

BY

Lt Col I. C. Narang*

The most frequent injury encountered by military surgeons during war, as they have always been, wounds caused by missiles, though as weapons of war have changed through the ages, the wounding effects which they are able to produce have undergone corresponding changes.

The behavior of the missile upon its penetration of the body is influenced by the density of the tissue injured and the elasticity of the skin and other tissue as well as by the nature, size and shape of the missile and its velocity, kinectic energy and stability at the time of impact.

Foreign bodies may be removed by immediate operation (Primary removal) or by intervention at some later period (Secondary or delayed removal) or left alone if embedded deep in the tissue and not likely to cause any harm.

In primary operation, it is neither possible nor advisable to adopt the complicated methods of localisation or procedure which may be indicated in secondary operations.

When for one reason or other a foreign body remains in the body, its removal may be indicated for various reasons :—

(a) Its presence may prevent the wound from healing.

(b) It may be responsible for recrudescence of inflammation.

(c) It may be causing pain or be interfering with function.

(d) The knowledge of its continued presence in the body may be the cause of mental or psychical symptoms.

In deciding whether or not to remove a projectile the foregoing consideration must be taken into account. It is always necessary to ascertain as definitely as possible, the position of the soldier at wounding, since his position has much to do with the location of the retained bullet which might have taken a bizare course and finally lodged in most unlikely place having no relationship with the wound of entry. Up-to-date accurate pre-operative radiological localisation is essential as it reduces the time for removal to a minimum.

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Occasional cases are encountered in which a missile has entered the open mouth and buried itself in the tongue or floor of mouth. Recently one such bullet has been removed by me from a soldier after 2 years of the injury which was embedded in the anterior 1/3 of the tongue. This was not detected by the hospitals where he was attended to, during the active phase of his treatment and the patient himself was oblivious of its presence but for his inability to protrude the tongue fully, difficulty in eating and distorted articulation.

This patient, a Gorkha Soldier of 4/5 GR, received a bullet injury on 4.12.71 in the Eastern Sector during the last Indo-Pak war. The bullet entered his oral cavity through the left angle of the mouth causing soft tissue injury at the site of entry, destroyed two teeth and produced a small wound in the tip of his tongue, (Fig. 1). He was treated in various hospitals in the channel of evacuation and finally landed in a Command Hospital where his angle of mouth was repaired.

He was unable to protrude out his tongue fully. His initial difficulty in eating however, gradually disappeared but distorted speech and sense of tightness in the anterior portion of the tongue persisted, which was taken lightly both by the soldier as well as his RMO.

However, after a lapse of 2 years, he was referred to SOPD with the above complaints on 17.10.73. On local examination a firm mass of the size of a small lemon with a hard core was felt in the substance of anterior portion of the tongue, bulging mostly towards the under surface in front of frenulum. Mucosa all around was intact except at one site where a small circular brownish patch was noticed.

Diagnosis of retained FB with surrounding reactionary fibrosis was made. Next day under GA through a direct incision over the mass, full bullet (Fig. 2) weighing 8.5 grams was taken out. Patient had full recovery within a week. At present he can speak clearly and the tongue has become more mobile.

Anterior portion of the tongue, where bullet was found embedded, is highly mobile part and it is used all the time while talking and eating. Surprisingly, in such delicate and active part, the patient carried a full bullet and its presence was never suspected for almost 2 years. Probably it was taken for granted both by the patient and the attending doctors that bullet after entry must have had passed out throught the open mouth and

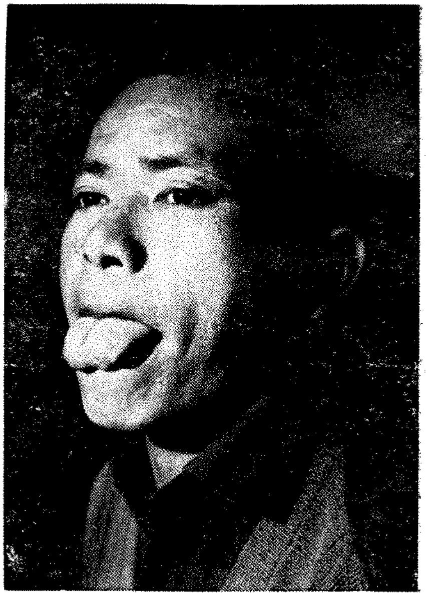

Fig. 1

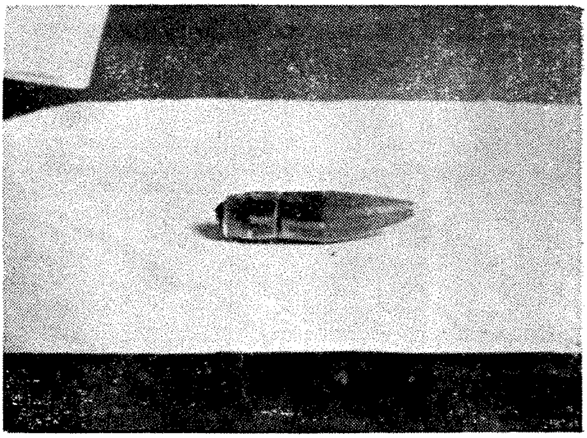

Fig. 2

consequently, nobody looked for it. When the bullet was shown to the patient, he was very much surprised and now he is keeping it as a souvenir.

I thank Colonel SS Dhanoa, OC MH Dehradun for allowing me to publish this case.

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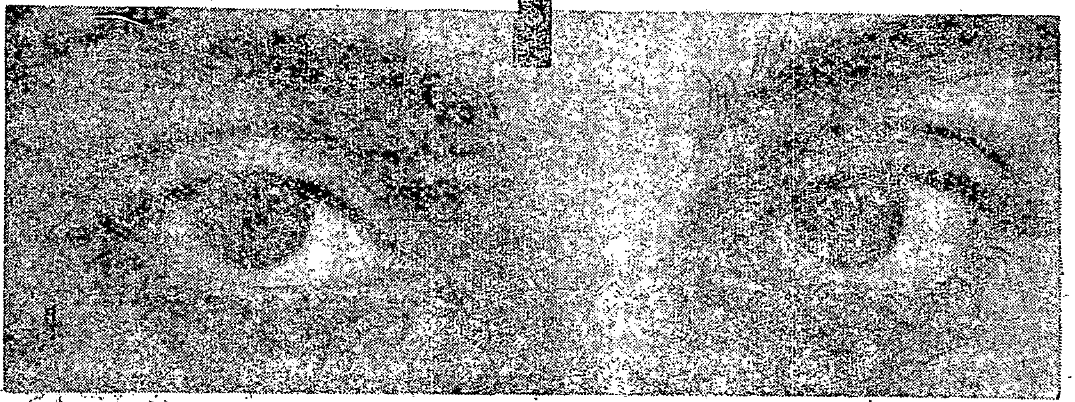

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MANAGEMENT OF DEGLOVING INJURY THUMB

BY

Maj Ajaipal Singh* & Lt Col P. S. Shukla**

INTRODUCTION

Function of the hand has been essential since the humanity began. Hand provides liberty and independence and may be the only fortune of a manual worker. Hand injuries are the most frequent occupational accidents and account for the largest share of the cost of treatment, indemnity and pension and its initial mismanagement, often accounts for considerable loss to the society.

The ability of apposition of thumb resulting in grasping mechanism combining great strength and finely controlled accuracy differentiates human hand from manus of other animals.

The aim of this short paper is to document the management of a case of degloved injury of right thumb treated in a peripheral hospital with restoration of complete function and form.

CASE REPORT

Hav SPS reported to this hospital on 4.5.73 with degloving injury of right thumb due to the thumb having been caught in the moving belt of a thresher the same day. The entire skin from the base of the thumb was avulsed, completely denuding the thumb with the tendons and small muscles intact (Fig-1). Management comprised of three phases.

Stage I (4.5.73)

Under general anaesthesia, the denuded thumb was burried in a subcutaneous tunnel in the right lower quadrant of the abdomen, the margins of the tunnel were accurately sutured to the cuff of the skin at the base of the thumb. The mobility of the thumb in the subcutaneous plane was restricted by two suitably placed mattress sutures through the overlying skin. The thumb was strapped to the donor site leaving the fingers free for vigorous active movements.

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** Classified Specialist (Surgery), Military Hospital, Meerut.

Stage II (25.5.73)

Sutures were removed on the 10th postoperative day and the healing was firm by primary intention. The thumb was kept buried in a tubed pedicle flap raised from the abdomen. The seam of the tube was along the radial border of the thumb. The abdominal skin defect was closed by extensive undermining and primary closure with tension sutures leaving a drain through a separate stab wound at the dependent point (Fig-2).

Stage III (8.6.73)

The thumb with the tubed pedicle flap was released from the abdomen. Any redundant skin was trimmed and reshaped and after excision of the nail bed was finally closed at the tip. The residual abdominal skin defect was again closed after undermining and primary suture.

At all stages, the wounds healed by primary intention and the patient regained full movements of the metacarpophalangeal and interphalangeal joints (Fig-3).

DISCUSSION

The function of the hand is of such great importance that utmost care and skill must be exercised in the management of all hand injuries. Neglect of even minor injuries can have so serious a result that the present trend is to treat them in specially organised clinics. In the absence of such centres, majority of these cases fall to the lot of general surgeons and it is the initial treatment that decides the final functional recovery.

Barring tendon injuries and compound fractures, all injuries involving extensive loss of skin in the hand and fingers should be treated by early, adequate and lasting skin graft in order to avoid contractures of tendons and capsule due to exposure and ischaemia. This hastens healing, prevents sepsis and by obviating fibrosis and contractures preserves function of the deeper structures.

Partial thickness grafts are less satisfactory but may be used as a temporary expedients but as far as possible, full thickness cover should be attempted as a primary or delayed elective procedure. Loss of skin from the body of the hand or large part of the thumb usually necessitates a pedicle graft from some other site like; abdomen, thigh or opposite arm. As a primary measure, this may present unexpected difficulties even when undertaken under ideal conditions except for those with some experience of plastic surgery. The uncertainity of asepsis, troublesome bleeding and difficulty of obtaining adequate immobilisation of the injured hand against the donor site will militate against the success of a graft.

In cases of severely denuded single finger when the blood supply of the deeper structures have been seriously depleted (ring avulsion) adoptation of amputation as a primary measure is recommended but such drastic decision cannot be entertained for similar injuries of the thumb.

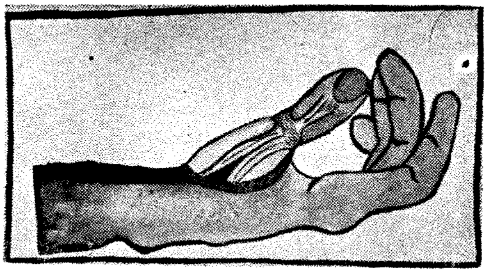

Fig. 1

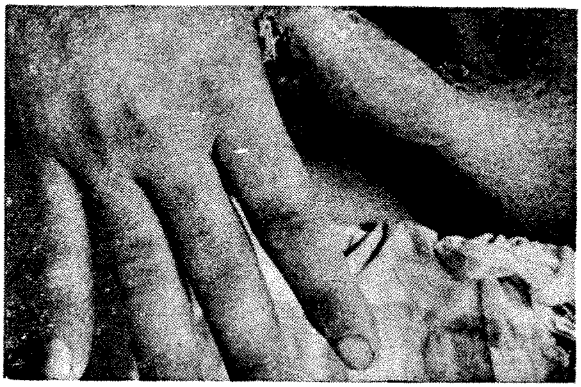

Fig. 2

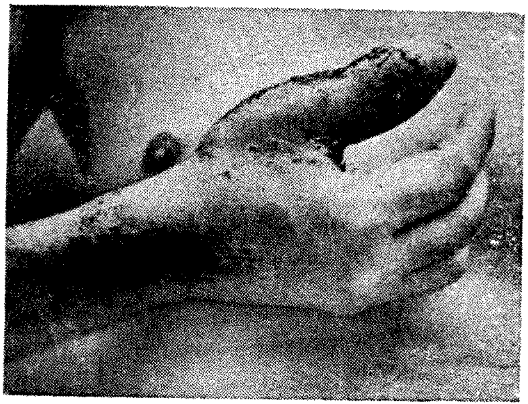

Fig. 3

Thumb is equivalent to about half of the hand and a rigid stump of the thumb is better than no thumb at all. It is indeed difficult to overemphasise the importance of avoiding primary amputation of the thumb.

SUMMARY

The management of a case of degloving injury of the thumb has been presented. The principles of treatment of hand injuries have been briefly discussed.

ACKNOWLEDGEMENTS

We are grateful to Col D Ramesh Rao, Commanding Officer, Military Hospital Meerut for his kind permission to report this case.

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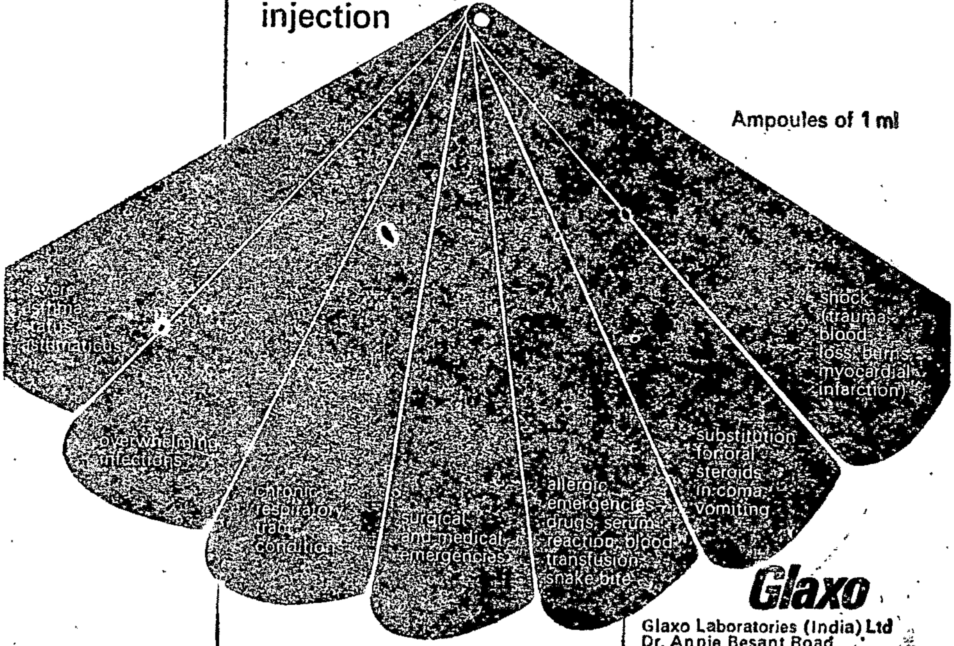
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3 BROTHERS

BILATERAL CYSTIC HYGROMA NECK

(An Anaesthetic and Surgical Problem)

BY

Capt Yudh Vir Suri* Col B.R. Baliga**
Brig A.C. Bose*** Maj J. Kar****

INTRODUCTION

Cystic hygroma are the congenital malformations of the lymphatic system rather than true neo-plasms. These tumors are the spaces lined by the endothelial cells, containing lymph arising from sequastrated lymphatic tissue which fails to link up with the main lymphatic system. They may be small in size or large cyst-like spaces usually multiloculated which may be several inches in diameter. These tumors are usually benign and may be small and well localised or diffuse infiltrating in nature. Approximately 50-60% of these lesions are present at birth and 80-90% are detected before the end of the second year of life.

The tumor occurs in two forms, lymphangiomatous variety and cystic hygroma. Those found in the neck, axilla, inguinal, retroperitoneal and sacral regions are predominantly cystic hygromas, whereas those of the tongue, face, skin, mucous membrane are almost of the lymphangiomatous variety.

In the neck hygroma colli, in the most common type of cystic lymphangioma. It is usually demonstrable at birth or any time during the first two years of life, as a soft poorly defined mass, often in the posterior cervical triangle. Many of the tumours show trans-illumination. Rarely it may occupy the anterior triangle, usually high up under the mandible. It may extend into the floor of the mouth, base of the tongue, pharynx, larynx covering the vocal cords.

The tumour may surround vessels and nerves, making surgical excision difficult. A sudden and rapid increase in size of the tumour may occur at any time due to haemorrhage into the cystic cavity or infection into the drainage area.

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Harrison and Johnson have reported a case of lymphangioma of the tonsil while Stadler et al (1952) and Shaw (1965) have each reported a case of lymphangioma in the retro-pharyngeal tissue. Narang and Mehta (1966) reported a case of lymphangioma larynx with post-operative respiratory obstruction.

A careful review of literature has not revealed any case of bilateral cystic hygroma of the neck, covering bilaterally anterior triangle, posterior triangle down to the Supra-sternal notch, as found in the following case. It is presented as an anaesthetic and a surgical problem.

CASE REPORT

R. K., 3 years old male child, was admitted in Paed : Ward on 11.9.73 with a gradually increasing swelling since birth on both sides of the neck. It was quite painless and did not cause much disturbance to the child.

The parents mentioned that the child is having noisy breathing and difficulty in swallowing. Breathing difficulty is more when the child is lying flat on the bed. Breathing difficulty is relieved when the patient is lying with face down position. Swallowing difficulty is increasing as the swelling is increasing in size.

Patient was born normally after full term pregnancy. The various mile-stones had been quite normal.

On Examination

Child was quite active and cheerful and fairly well nourished. There was diffuse swelling on both sides of the neck, extending from submandibular region just above the clavicle (Fig. 1).

The tongue and oral cavity was thoroughly examined; tongue was of bigger size & flabby in character but there was no lymphangiomatous growth over the tongue, floor of the mouth and the pharynx.

The skin over the swelling was stretched. Mass was soft with ill-defined margins. It was non-tender and fluctuations were present. Trans-illumination was positive on right side. The patient had marked inspiratory stridor and position of the trachea could not be ascertained due to the swelling. However, there was no evidence of respiratory insufficiency.

Heart showed grade 3/6 murmur over the apex and right parasternal region. Clinically no organic lesion of the heart was detected. However, Ba-swallow was advised but could not be done as the child was not co-operative.

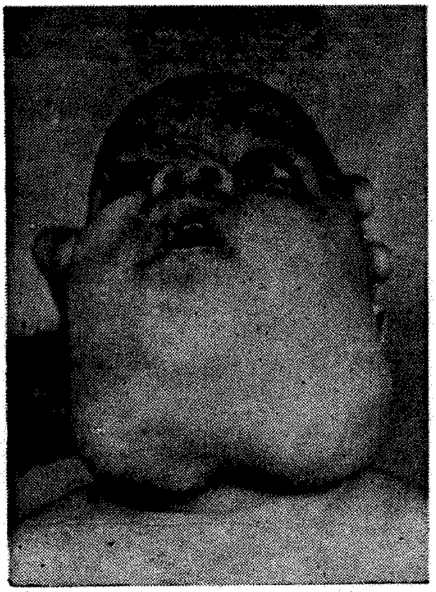

Fig. 1

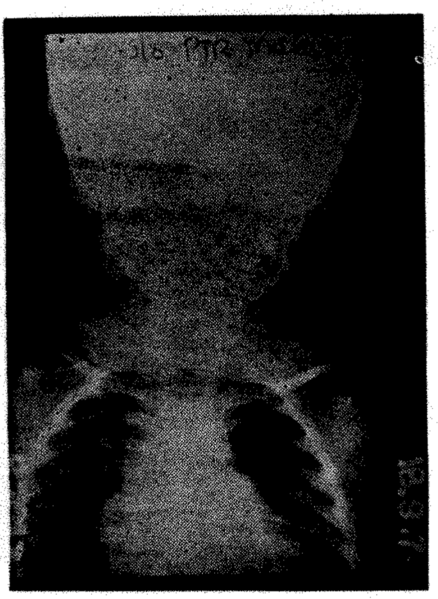

Fig. 2

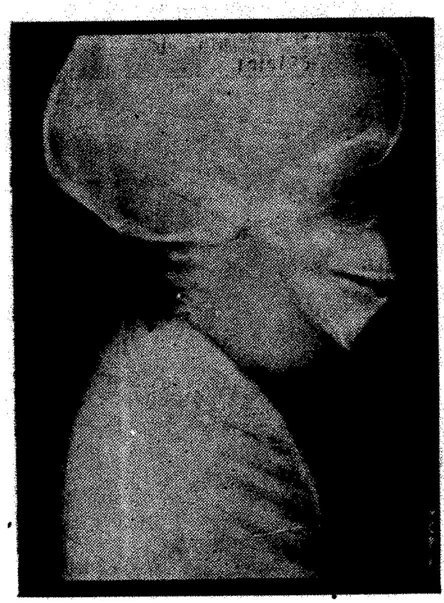

Fig. 3

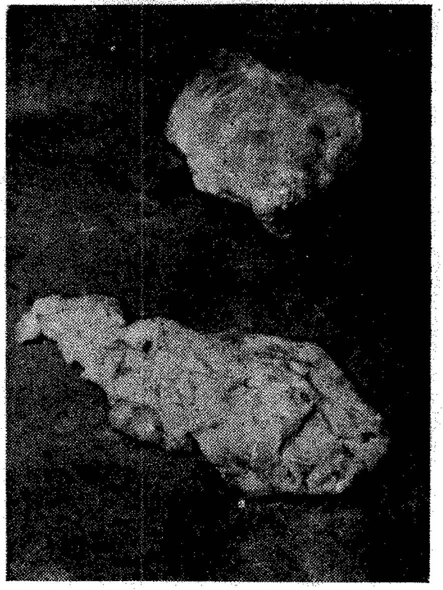

Fig. 4 Excised Mass.

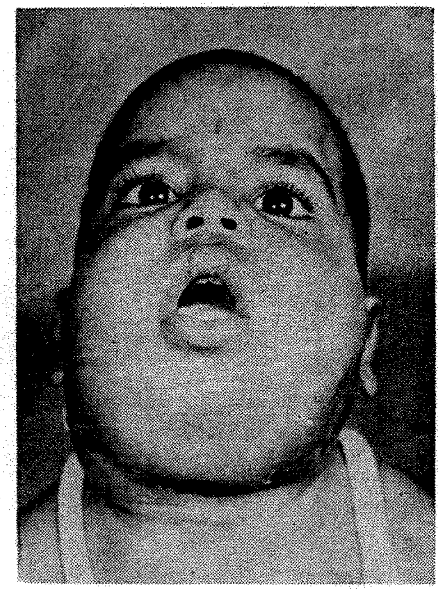

Fig. 5 Child after Operation.

Investigations :

Routine urine and blood examination were within normal limits. X–ray of the neck and chest showed large bilobular shaped homogenous cystic masses on either side of neck. (Fig. 2–3) A clinical diagnosis of cystic hygroma was made and it was thought that the stridor was probably due to pressure effect on the trachea.

Operative notes and anaesthesia

Child was operated on 26.9.73. As the child was well nourished vene–section was done before induction for replacement of blood loss. Anaesthesia was induced with nitrous oxide, oxygen and halothane through the Boyle's semi–open circuit and maintained with nitrous oxide, oxygen and halothane (0.5–1 %). Intubation was done with difficulty, with the proper sized endo–tracheal tube. The course of anaesthesia was smooth and uneventful during the operation. 200 ml of blood loss was replaced during the operation. The tumour consisted of cysts of various sizes which contained clear fluid. There was ramifications of the mass in between the vital structures in the neck which made it impossible to do the total excision. However, most of the mass was excised and the child stood the operation well. (Fig. 4-5).

DISCUSSION

Operative excision is the treatment of choice for these lesions and the only effective treatment available. Age is not a contra–indication to operation and the only exception may be the pre–mature infant in whom the operation may be done after the child attains the normal weight. In addition to the paediatric anaesthesia problems, the following points are to be considered :

1 Emergency tracheostomy is not possible to perform through the huge cystic hygroma in the neck if the need arises.

2 Intubation is difficult due to poor visualization of the larynx. Partial aspiration of the cyst should be done in suitable cases to perform intubation. Pharynx should be packed to avoid aspiration in case the tumor bursts into the mouth.

3 In case normal intubation fails a wide bore needle may be introduced into the trachea through the neck and passing a probe or catheter upwards and threading the tube through nose or mouth and then the tube is guided on the probe or catheter.

4 Intra–venous infusion should be set up before commencement of surgery and blood loss should be adequately replaced as we did.

5 All cases of cystic hygroma of the neck should have X–ray of the chest to exclude any mediastinal extension which may shift the trachea.

6. Traction of movements of the tumour by the surgeon will aggravate the respiratory embarassment.

7. Post-operative care of the patient cannot be over emphasised. Immediately after ex-tubation respiratory obstruction must be carefully watched which may be due to extension of the tumour over the larynx or due to reactionary oedema or collection of haemorrhagic fluid into the wound or floppy tongue due to damage to the hypoglossal nerve during surgery.

SUMMARY

A rare case of bilateral cystic hygroma of the neck is presented. The problems of difficult intubation and its management is described.

ACKNOWLEDGMENTS

We are grateful to Col AB Ray, Commanding Officer, Command Hospital, Central Command to permit us to publish this case from the Hospital records.

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ORAL HEALTH STATUS AMONG INDIAN TROOPS

BY

Brig M.D. Mehra* Surg Cdr (D) S.N. Luthra Maj S.K. Arora*****

INTRODUCTION

Epidemiological studies of rural and urban population in India to assess the oral health status and prevalance of dental disorders have been carried out by various workers. Reports are also available of such studies amongst Indian troops. A soldier, who is mainly drawn from rural population, is subjected to environmental and dietary changes in addition to regular dental care. Inspite of his varying back ground, he conforms to an intergrated pattern of living common to his socio-economic status in the service. This study has been undertaken to assess the oral health status and ascertain the different methods of maintaining oral health employed by the soldier.

MATERIAL AND METHOD

The present study has been carried out during 1973. The random sample comprised 1050 serving soldiers and recruits of different age groups from various units in Delhi Cantt. The examination included recording of personal data such as age, habits like smoking, tobacco, pan and betal nut chewing, along with method and material used for cleaning the teeth. Clinical examination was carried out under a good and uniform operating light using mouth mirror and probe. Oral hygiene index and periodontal status was determined according to norms laid down by Greene and Russel respectively.

Table I

Age wise distribution of cases examined

Age Group	Number of peronnel examined
18 – 25 Years	416
26 – 33 Years	459
34 – 41 Years	157
42 Years and above	18
	1050

---

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Table I shows the number of men examined and their age groups. Being a random sample, information in respect of age group 42 years and above under all tables is not considered statistically significant due to the small size of the sample.

Table II

Percentage distribution of cases showing personal habits

Age Group	Number examined	Smoking	Tobacco Chewing	Pan and betal nut chewing
18–25 Years	416	49.4%	1.2%	0.5%
26–33 Years	459	64.5%	2.6%	2.8%
34–41 Years	157	69.4%	2.5%	4.5%
42 Years and above	18	61.1%	5.6%	11.1%

This table shows general rise in personal habits with increase in age and service. Smoking among troops rises with advance in age and service; similarly tobacco chewing and pan and betal habits rise, though to a small extent.

TABLE III

Percentage distribution of cases showing methods and materials used for cleaning teeth

Age Group	Number examined	Method			Material		
		Datun	Tooth brush	Finger	Tooth paste	Tooth powder	None
18–25 Years	416	8.2%	90.4%	1.4%	89.7%	2.2%	8.2%
26–33 Years	459	3.7%	94.8%	1.5%	91.9%	4.4%	3.7%
34–41 Years	157	6.4%	90.5%	3.2%	86.6%	7%	6.4%
42 Years and above	18	0%	94.4%	5.5%	83.3%	16.7%	0%

This table shows the methods and materials used by service men for cleaning their teeth. Recruits on joining are introduced to tooth brush in training centres and 90.4% in age group 18–25 years are using tooth brush with tooth paste or powder for cleaning the teeth and this is continued in other age groups as well. Datun users do not use any other material.

Table IV

Percentage distribution showing oral hygiene (Greene's Score)

Age Group	Number examined		O	H	I
		O	I	II	III
18–25 Years	416	8.7%	86.3%	5.1%	0%
26–33 Years	459	5.7%	82.6%	10.6%	1.09%
34–41 Years	157	10.8%	75.8%	12.1%	1.3%
42 Years and above	18	5.5%	72.2%	22.2%	0%

Note:– **OHI Score**

O denotes absence of calculus (tartar deposits) and good oral hygiene.

I denotes presence of supra gingival calculus deposits (Marginal only) and dental plaque.

II denotes deposits of calculus covering tooth surface (fair amount)

III denotes gross presence of calculus deposits (supra and sub gingival). Neglected oral hygiene.

Table IV, shows the oral hygiene index. It is seen that the healthy condition prevails at 8.7% and rises upto 10.8% with methods used for maintaining oral hygiene and dental care in age groups 18–25 and 34–41 respectively.

The oral hygiene index I denotes the presence of calculus deposits and dental plaque which is high at 86.3% and drops approximately by 10% in higher age groups, as a result of being inducted to dental care and education in maintaining good oral hygiene on joining service. The oral hygiene index II which denotes fair amount of calculus deposits shows slow rise in the percentage from 5.1% to 12.1% in the same age groups. This may be attributable to lack of routine dental care.

Table V

Percentage distribution showing periodontal disease (Russel's Score)

Age Groups	Number examined	Periodontal Status O	1	2	6	8
18–25 Yrs	416	55.8%	39.7%	4.6%	0%	0%
26–33 Yrs	459	38.1%	50.1%	8.5%	2.0%	1.3%
34–41 Yrs	157	21.0%	65.6%	12.1%	1.3%	0%
42 Years and above	18	16.7%	50%	33.3%	0%	0%

Note – Russel's Score.

0 denotes normal
1 Mild gingivitis.
2 Generalized gingivitis and early periodontitis.
6 Periodontitis, periodontal pocket formation and alveolar bone involvement.
8 Advanced periodontitis with deep periodontal pockets.

Table V shows the periodontal status. The presence of early periodontal condition like gingivitis (Russel's Score I) is present at 39.7% in 18–25 age groups, this rises to 65.6% with advance in age.

The evaluation under Russel's score 2 and 6 shows that the condition progresses with rise in age. Russel's score 2 being 4.6% to 12.1% and 33.3% in 42 years and above age group.

DISCUSSION

This sample survey to evaluate the oral health status habits and methods to maintain oral hygiene has been carried out among troops and its co-relation to periodontal disease established.

In 18–25 years age group 90.4% of the troops use tooth brush and tooth paste/powder for maintenance of oral hygiene and 6.4% of the total 1050 personnel continue to use datun or finger for cleaning teeth and gums.

From oral hygiene index based on Greene's score for evaluation, it is seen that only 8.7%, 5.7% and 10.8% in 18–25, 26–33 and 34–41 age groups respectively maintain good oral health. The others though using the same method, show a high percentage of dental plaque and marginal deposits of salivary calculus.

This when co-related to periodontal disease shows healthy gum condition in 55.8%, 38.1% and 21% amongst the same age groups–Russel's Score 0 and rise in periodontal disease as marginal gingivitis from 39.7% to 65.6% under Russel's periodontal score 1 for the same age groups.

The periodontal disease, though high in India population, is less among the troops who are under regular dental care and take to tooth brushing and tooth paste to maintain oral hygiene. Luthra (1970) found the incidence of periodontal disease among service personnel and families in similar age group at 71.6% rising to 86.6%.

Mehra et al (1966) gives the oral hygiene index for 10.8% as good 64.0% as fair and 25.2% as poor and prevelance of periodontal disease at 71% amongst Army, Navy and Civilian personnel.

Ramachandran et al (1973) in his studies of civil population who use variant methods to maintain oral hygiene finds incidence at 95.5% in rural and urban population of Tamil Nadu.

Although the finding of other workers is similarly high, it is seen from this sample survey of service personnel that early induction to habit of maintaining oral hygiene and regular dental care is paying dividends as to the incidence of periodontal disease amongst troops which rises only upto 65.6% in age group 33–41 years. It also shows that periodontal disease, if allowed to advance, is destructive to the periodontal tissue but can be controlled with regular dental office and home care. The advance of periodontal disease to Russel's score 2 – generalised gingivitis or early periodontitis, is explainable when co-related to oral hygiene index I which shows presence of dental plaque and supra gingival calculus.

SUMMARY AND CONCLUSION

90.4% of service men 18–25 years age group use tooth brush with tooth paste/ powder to maintain oral hygiene while the remainder depend on datun and finger massage. Oral hygiene index shows healthy condition in 8.7% in 18–25 age group and rises to 10.8% in 34–41 age group. OHI score I at 86.3% in 18–25 age group drops to 75.8% and 72.2% in 34–41 and 42 and above age group, as a result of dental care and education in maintaining good oral hygiene. The incidence of periodontal disease which is on everage of 71.6% to 86.6% among service personnel and 95.5% in urban and rural Indian population of Tamil Nadu shows an improvement in the sample survey under Russel's score '0' 55.8% and score I 39.7% in age group 18–25 which rises to

65.6% in 34–41 age group. Thus a better oral health status can be achieved by adequate motivation and dental health education in office and home dental care.

ACKNOWLEDGEMENT

We thank Major General RN Dogra, PVSM, PHDS, Director Dental Services for his valuable advice and guidance in carrying out study of the problem among service personnel.

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FABRICATION OF CAST ALUMINIUM BASES IN COMPLETE DENTURES

BY

Lt Col O.P. Joneja,* Brig. M.D. Mehra, **
Maj Gen R.N. Dogra, PVSM, PHDS. ***

Complete denture bases have been cast in aluminium way back in 1860 by Dr. J.B. Bean of Baltimore[1]. Metal bases are extensively used in developed countries for fabrication of partial dentures and in selected complete denture cases. The metals commonly used for fabricating such dentures are gold and chromium cobalt alloys which are expensive.

A well cast non porous metal base can be kept in a more hygienic and cleaner state. In addition, it provides necessary stimulation to the tissues due to better thermal conductivity as compared to methyl methacrylate denture bases. The tissues take more kindly[9] to metal bases, whereas, according to Anthony and Peyton[2], retention, health or stability of tissues may be affected when using acrylic bases due to inaccuracies developed during their processing.

In 1963 Lundquist[5] stated that cast aluminium test dentures with poorest adaptation in the palatal region had less discrepancy than the best fitting acrylic test dentures. Swartz[8] in 1966 found that the aluminium bases can be cast to fit oral tissues and provide proportionately better retention than other base materials, thus supporting Lundquist's findings.

Investigations and clinical findings of 180 complete denture cases at A.F.M.C. with anodised aluminium bases have proved the superiority of these bases over the acrylic dentures in regard to tissue health and retention thus corrobrating the earlier evidence.

---

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**Command Dental Adviser Western Command (Formerly Professor and Head of Department of Dental Surgery, Armed Forces Medical College, Pune-1).

***Director Dental Services, (Formerly Professor and Head of Department of Dental Surgery, Armed Forces Medical College, Pune-1).

Anodised aluminium bases are quite stable in oral fluids and no pitting or disintegration has been observed clinically during review over a period of three years. Slight discolouration of bases was, however noticed in a few cases but hyperplasia or toxicity of any kind of the tissues was absent.

Histological study of stratum corneum in some of these cases has shown that average thickness of stratum corneum has been found to be greater under aluminium base dentures when compared to response under routine acrylic dentures. Cytological observations also confirmed the histological findings. It appears there is a stimulation of keratinization under aluminium base dentures as evidenced by a higher keratinization index under such dentures[3,7].

Some of the important criteria for selection of denture base material are accuracy of fit, tissue tolerance, light weight and high thermal conductivity[6]. These requirements are ideally met by aluminium which is inexpensive and readily available. Inspite of this, the metal is not being used by the profession. It is difficult to understand the reasons for this attitude because the metal can be cast with ordinary casting equipment. However, an exacting technique is essential to ensure porosity free casting as there is a high setting contraction during this procedure. In order to avoid voids specially in the palate region the following technique which has given consistently good results must be followed.

PROCEDURE FOR ALUMINIUM CASTING

Base Coverage outline

Survey the master cast and extend the outline of the cast upto the height of contour on the facial side of the ridge to avoid engagement of any undercut by the cast metal base.

Waxing and Sprueing

1 Wax the cast with combined thickness of No. 8 & 6 @ casting sheet wax.

2 Provide lingual finishing line by adapting No 6,½ round inverted wax form over the palatal wax. Using wax carver, create slight undercut towards the facial wax.

3 Attach 20 gauge round wax loops to the wax sheet over the residual ridge at an interval of about 8 to 10 mm, with sufficient space beneath to form the attachment for acrylic resin denture base (Fig 1).

@ Ash Solbrig shaped sheet wax. Amalgamated Dental Trade Distributors, Ltd., London.

4 Sprue the wax pattern. Multiple oversized sprue formers, 4 mm in diameter, are applied in the areas as shown (Fig 2), to compensate for high setting contraction of metal. These sprue formers are joined to a big reservoir from where three sprue formers of 1.5 to 2mm in diameter, open independently towards the crucible former. In addition each oversized sprue former should have reservoir placed not more than 3 mm from the pattern.

Investing

Invest the wax pattern in sterling* or any other acceptable investment using double investment technique.

Burn out

1 Heat slowly to 700°C in 60 minutes and heat soak the ring for 30 minutes.

2 Switch off and cool the ring to 300–370°C in the furnace before casting, as the casting shrinkage decreases at lower mould temperature.

Casting

1 Melt but do not over heat the metal before casting. Aluminium neither exhibits a mirror like surface nor spins like gold alloy when fused. No attempt should be made to obtain these conditions in melting aluminium. Aluminium, when melted[1] settles upon the floor of the crucible, although its upper surface remains spheroidal. Cast[4] as soon as the metal becomes molten to prevent in–corporation of hydrogen.

2 Use flux and skim off the oxide formed before casting as fluxing cleanses and degases the molten metal.

3 A suitable flux must be used. Eutector£ flux gives good results as borax is unsuitable.

Heat treatment

1 Bench cool the ring to room temperature.

2 Do not quench.

Finishing

1 Cut off the sprues with separating discs, fissure burs, with light pressure as close to the casting as possible without damage to the metal base (Fig 3).

---

* *Engelhard Industries Ltd., Sutton Surrey, England.*

£ *Eutector Flux 190, Larsen and Toubro, Bombay.*

2 Grind off remaining portions of sprues so as to blend them with the metal base with heatless stones.

3 Smoothen the coarse surface with green alpine stones of finer grit.

4 Remove fins if any, either with No. ½ or No. 1 round bur or with green stone of finer grit of appropriate size.

5 Place aluminium base on the duplicate cast and remove scratches if any, with fine sand paper discs and rubber wheels. Polish the external surface of the aluminium base with fine pumice and soft brush, using medium pressure (Figs. 4&5).

6 Finally, remove the metal base from the duplicate cast, and wash with soap and water to remove all traces of polishing material.

CONCLUSIONS

1 A detailed technique has been given to ensure acceptable casting of aluminium denture bases. This has proved successful in training Dental Technicians at the Dept of Dental Surgery at A.F.M.C. Pune.

2 Aluminium is inexpensive and readily available and requires no special equipment and materials for its casting.

3 Complete dentures with aluminium bases are superior to acrylic dentures for tissue health and give better retention. These are specially indicated to secure better retention in difficult cases with marked alveolar resorption.

4 The cast aluminium bases must be anodised. Anodising forms a protective oxide film to prevent corrosion.

5 A suitable alternative to expensive dentures in chromium cobalt or gold alloys is presented.

ACKNOWLEDGEMENT

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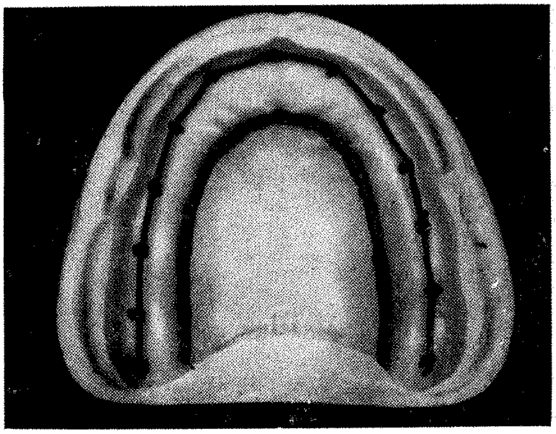

Fig. 1 Wax pattern showing lingual finishing line and wax loops In for retention of acrylic resin.

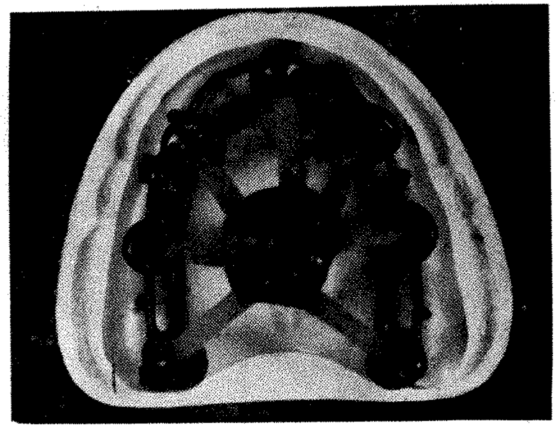

Fig. 2 Wax pattern with multple over sized sprues to compensate In for shrinkage of the metal.

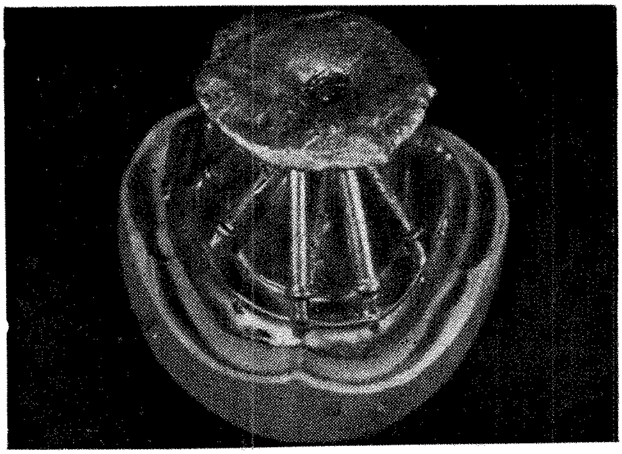

Fig. 3 An aluminium casting.

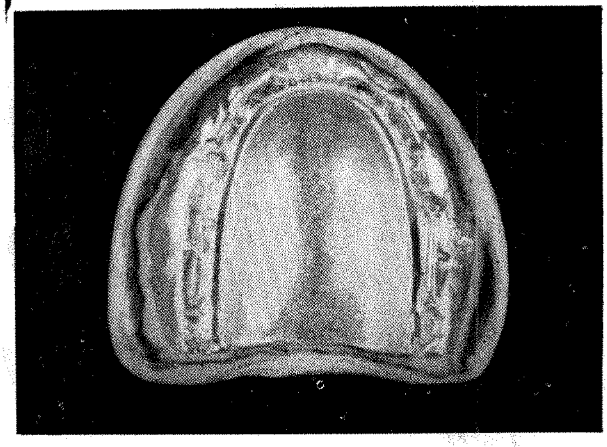

Fig. 4 View of the external surface of the aluminium denture base on the master cast.

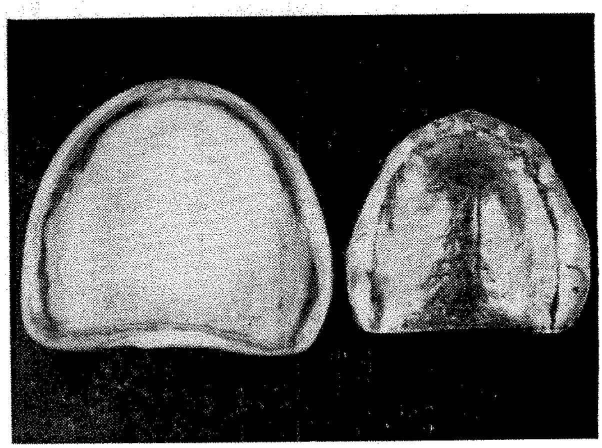

Fig. 5 View of the internal surface of the aluminium denture base by the side of the master cast.

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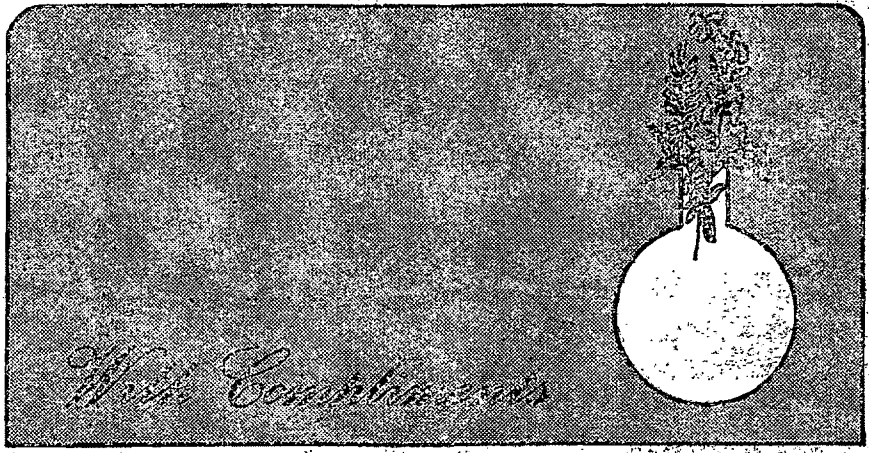

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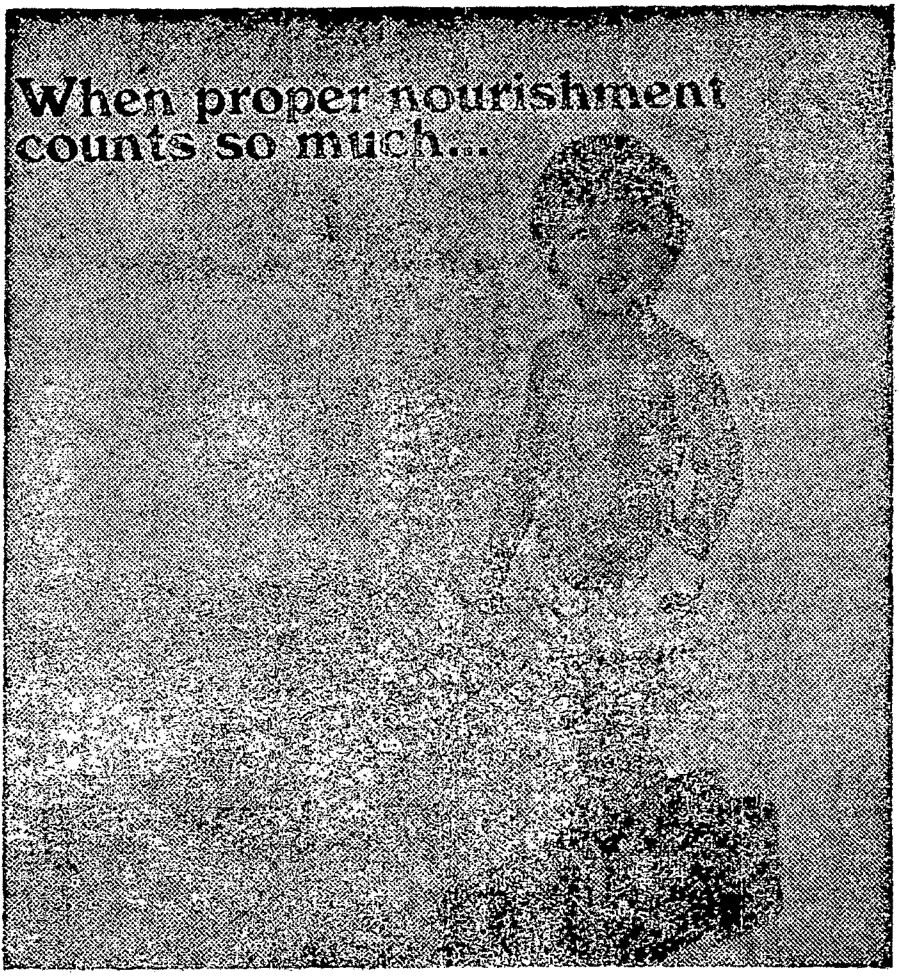
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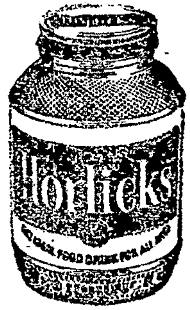

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ANODISING OF ALUMINIUM BASES FOR COMPLETE DENTURES

BY

Lt Col O. P. Joneja*, Brig. M. D. Mehra,**
Maj Gen R. N. Dogra, PVSM, PHDS.***

ANODISING OF ALUMINIUM BASES

Aluminium is a very readily oxidized metal. As a result of its affinity for oxygen, aluminium when exposed to air, forms a thin protective oxide film. This film is both adherent and relatively impermeable[2]. Any mechanical damage to this oxide film is self healing, because immediate reoxidation occurs[1].

This natural oxide film is sufficiently protective. It offers good resistance to corrosive attack in normal use, being resistant to water and atmospheric impurities. However, this thin natural oxide film is not adequate to protect the metal and it is advisable to build up an even thicker and more resistant film by electro-chemical means. Aluminium used electrically as an anode combines with the electrolytically produced oxygen to form aluminium oxide on the surface of the metal. It is possible in this way to build up much thicker and harder layers which offer greatly improved protection against corrosion than can be derived from simple oxidation. This electrolytic oxidation is called anodising and must be used for protecting aluminium dentures and appliances from various corrosive agents.

PREANODISING PROCEDURE

The cast aluminium denture base which is to be anodised, must be free from pitting; as this defect is not covered by anodising, Before anodising, the denture base must be thoroughly smoothened and cleaned so that the electrolyte can uniformly attack the metal.

The general procedure comprises:-

(a) Etching the object by immersing in NaOH.

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(b) Rinsing and finally neutralising with dilute nitric acid.

(c) Brightening procedure.

Etching

Etching is done by immersing the aluminium denture base in an unbreakable glass container containing 15% of commercial NaOH in water, at a temperature of 30 to 60°C for 30 seconds to 60 seconds. Etching more than the recommended duration will render the aluminium denture base unusable by excessive attack.

Rinsing and Neutralisation

Rinse thoroughly in running water and neutralise in 50% commercial nitric acid at a temperature of 30 to 70°C for 30 seconds in an unbreakable glass container. Thoroughly rinse again in running water.

Brightening procedure

The neutralised aluminium denture base can be chemically polished, by immersing in an unbreakable glass container containing phosphoric acid 96%, nitric acid 4% and nickel nitrate 0.2%, at temparature of 90°C for a maximum duration of 30 seconds. A variation of 10°C either way is acceptable. Thoroughly rinse again to avoid any possibility of contamination of the electrolyte from traces of brightening solution.

ANODISING PROCEDURE

20% sulphuric acid in water (approximately) with specific gravity of 1.145 is used as electrolyte[1]. The temperature of the electrolyte must be maintained at 18–20°C to avoid formation of undersirable powdery[2] film at higher temperature. Double container is used to control the temperature of the electrolyte which rises during anodising. The electrolyte is kept in an inner unbreakable glass container which is surrounded by ice in an outer metal container.

Electrical Data

DC voltage ranging between 12 to 20 volts and with a current density of 5.5–27 m.a/cm^2 at a temperature of 20°C is required. For an average upper denture about 2 amperes current is adequate.

Operation of Anodising

The aluminium denture base is made anode and is connected to the +ve terminal of anodising apparatus. The lead strip, about 2.5 cms × 14 cms, is made cathode and is connected to the –ve terminal of the apparatus (Fig 1).

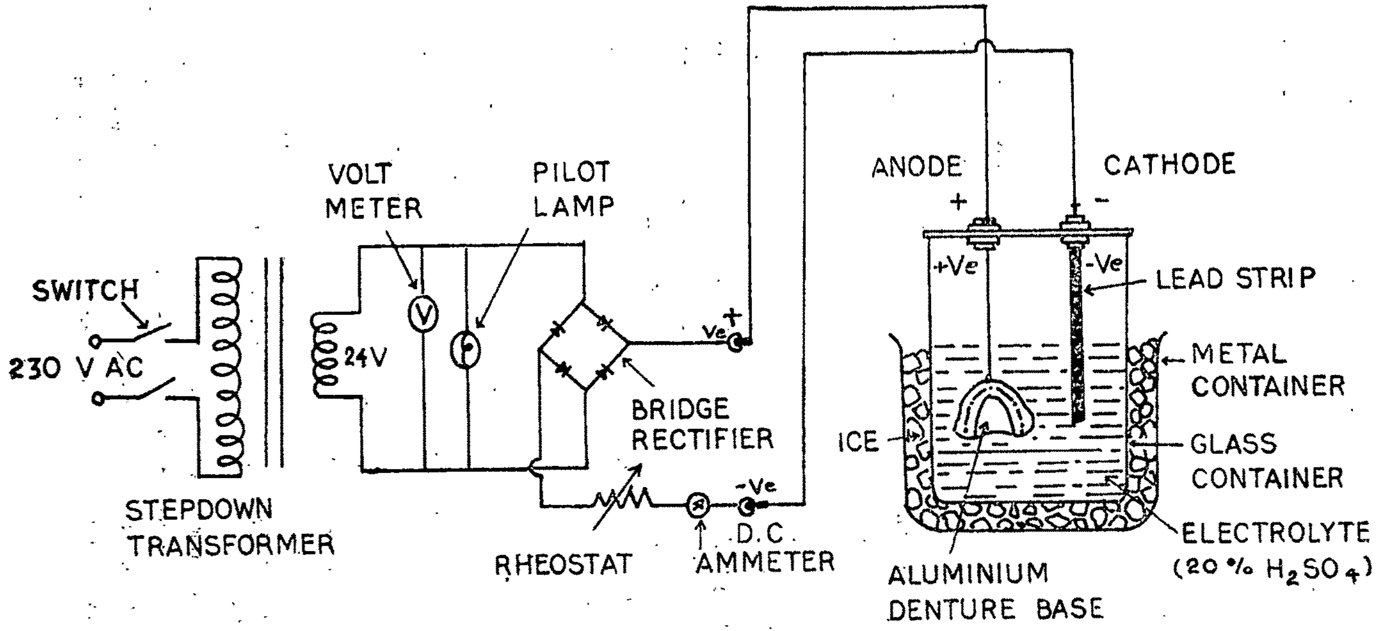

FIG. I - DIAGRAM OF THE ANODISING APPARTUS.

Anode and cathode are kept immersed 10 cms apart in electrolyte which is filled in unbreakable glass jar of approximately 15x 10x 20 cms and maintained at the desired temperature. The current is regulated by the rheostat and its value observed on the DC ammeter. The value of the current to be passed is according to the total surface area of the denture base for 30 minutes to 60 minutes. A 24 volt pilot lamp and a 0–30 volts range voltmeter are incorporated in the anodising apparatus.

The current is switched off when the anodising is completed and the aluminium denture base is removed from the electrolyte.

SEALING

The electrically produced oxide film is very absorbent immediately after its formation and if due care is not taken in thoroughly cleansing the denture base after its removal from the electrolyte, it may retain sulphuric acid. Aluminium denture base must be thoroughly washed in running water for 5 to 10 minutes and then kept in water for another 10 – 20 minutes. There will be slow diffusion of the acid from the porous structure of the oxide film. The water should be changed two or three times during this period.

The porous oxide film must be sealed. Sealing closes the pores and renders the oxide film resistant to corrosion. Inadequate or unsatisfactory sealed oxide film does not offer resistance to corrosion which is the aim of anodising. The sealing is carried out by suspending the item for immersion in boiling distilled water of pH 6–7, for a period of 30–60 minutes; in unbreakable glass container. After sealing is achieved the denture base is rinsed in running water and dried.

TEST OF ANODISING

One must be certain that the denture base has been properly anodised, so that it can offer good resistance to corrosive attack when used. Aluminium when anodised is electrically non conductor.

Whether a suitable layer of aluminium oxide has been formed on the denture base can be tested either with the help of a torch cell and a torch bulb or by use of an electric bell (Figs 2 & 3). The method of testing in each case is given below :—

(a) The negative of a torch cell is made to contact the denture base and the positive of the cell is kept in contact with one terminal of the bulb. The other terminal of the bulb is connected to the denture base with a wire. If the denture base has been properly anodised, no current will flow and hence the bulb will not light up.

(b) An electric bell is connected through a switch from the mains. The anodised denture base is included in the circuit with the help of connecting wires attached to crocodile clips to hold the denture base. The electric bell will not ring on closing the switch if the oxide film has been properly formed on the denture base. In order to ensure that the denture base has been anodised at all places, the crocodile clips may be connected to various points on the denture base.

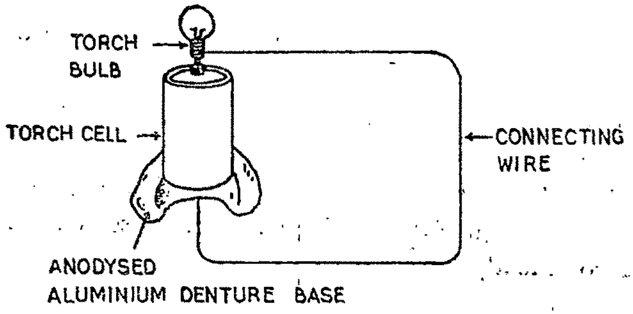

FIG. 2-TORCH CELL TEST: THE BULB WILL NOT LIGHT UP IN THE ABSENCE OF FLOW OF CURRENT.

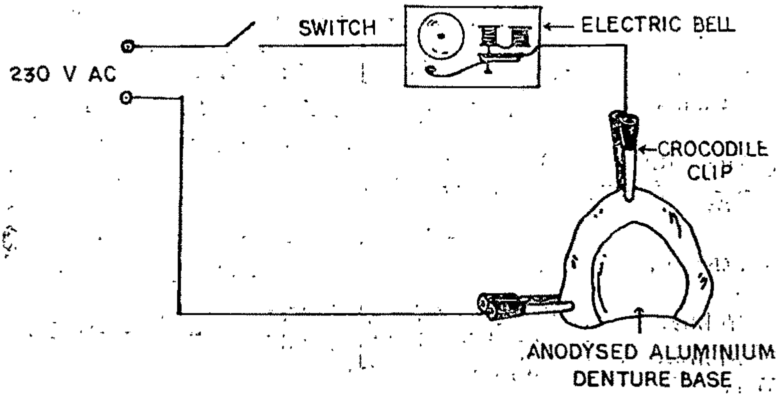

FIG. 3-ELECTRIC BELL TEST: THE ELECTRIC BELL WILL NOT RING ON CLOSING THE SWITCH.

SUMMARY

1 Anodising is electrolytic oxidation. The anodic film grows within the metal by conversion of aluminium to aluminium oxide.

2 The anodic film is extremely absorbent immediately after its formation and it must be sealed, as only after its sealing, it achieves high corrosive resistance.

3 The thickness of the film varies according to the duration of treatment. Greater the thickness, greater the resistance to corrosion.

CONCLUSION

Anodised aluminium dentures have a place in the management of edentulous cases. A step by step technique for anodising has been presented which if followed will ensure good results consistently.

ACKNOWLEDGEMENT

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The authors are specially grateful to Shri M L Arora, Assistant Technical Officer (Radiology) AFMSD Pune for providing circuit diagram of the anodising apparatus.

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FACIAL PAIN - REVIEW

BY

Maj BS Dahya*

One of the problems of dentistry is that treatment by medical practitioner and dental surgeon may overlap. Each should ensure, when it is relevent, that his colleague is fully consulted or informed. The meeting ground of medical and dental practitioners continues increasingly, to be the effects of dental treatment on patients receiving medication for systemic disorders. An anaesthetic in dental surgery or the carrying out of minor oral surgery may have serious consequences in patients receiving anti-coagulants or steroids. Similarly the medico – dental team is concerned with the effects of dental treatment in cardiac patients, general anaesthetics for dental patients and in the diagnosis and management of facial pains and a variety of other oral leisons with systemic etiology.

In this article the problem of "Facial Pain", will be reviewed from dental point of view.

'Dental disease is still the commonest cause of facial pain', - Henry Miller (1969). Many of us are unfortunately all too familiar with facial pain originating from dental sources, so that both young and old alike are often able to diagnose accurately for themselves the source of their discomfort, and even to indicate the offending tooth. It is not with this group of patients that I am concerned in this article, but with those whose facial pain originates from the less obvious dental lesion.

These patients are usually unaware of the dental origin of their pain and therefore present themselves to their doctor for relief. The doctor may reassure himself that the patient's pain does not originate in a dental source after a 'quick look round the mouth' with a wooden spatula and inadequate light. Such patients are then often passed through the hands of the otolaryngologist, physician, neurologist or neurosurgeon in an attempt to trace the source of their pain. They may be subjected to investigations which are unhelpful and at times even misleading. They may even be operated upon.

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*OC, 100 Dental Unit, Classified Specialist in Periodontology.

Since dental disease is so common it is desirable that any patient complaining of facial pain be examined by a dental surgeon whose clinical examination of the mouth should be supported by electrical and thermal vitality tests of the teeth and most important, radiographs of the teeth and jaws.

The minimal radiographic examination should include intra–oral films of the upper and lower jaw on the side involved and a posterior bite wing film of the teeth. A lateral oblique view film of the mandible is no substitute for good dental films.

A dental examination should include the inspection of every accessible surface of every tooth with dental mirror and a sharp probe under good light. The mandible, maxilla, floor of mouth, tongue and soft tissues and mandibular joints should also be examined.

PULPITIS

The tooth pulp is a well-innervated collection of blood vessels, lymphatics and connective tissue contained in a rigid chamber within the tooth. The nerve fibres within tooth pulp are peculiar in that no sensation other than pain can be elicited by their stimulation.

Acute pulpitis, invariably secondary to dental caries, is characterized by severe lancinating pain in the tooth and jaw, and although it may be precipitated by thermal changes it sometimes arises spontaneously. The pain often persists when the thermal change is removed. The patient may clasp his hand to his face during one of these paroxysms and approaches each meal with apprehension. If the inflammatory process spreads to the apical tissues the tooth becomes tender to percussion and this physical sign provides the diagnosis. Often, however, the tooth is not tender to percussion and these cases are likely to cause diagnostic difficulty if only a superficial examination is made.

Chronic pulpitis secondary to dental caries is characterized by less severe pain or may even be symptomless. If pain is present it tends to be intermittent and is usually described by the patient as a dull ache. The response to thermal change is much reduced.

Acute or chronic pulpitis may occasionally produce pain which mimics trigeminal neuralgia closely and this important differential diagnosis should not be forgotten.

The presence of a clinically sound restoration does not preclude the presence of dental caries. Recurrent dental caries can develop after a satisfactory restoration, or caries may have been incompletely removed at the time of cavity preparation (residual caries) by the dentist. In either case a dental radiograph supports or provides the diagnosis. It should be re-emphasized with regard to radiography of posterior teeth that the bite wing film is a sine-qua-non in detecting interproximal caries and should be taken in addition to peri-apical view of the teeth. The diagnosis can be made with the greatest of ease with a bite wing film.

Case 1 :- 38 year-old female patient, complaining of left facial pain for four months, diagnosed as trigeminal neuralgia, was examined by a dentist. The pain was intense and paroxysmal, limited to the maxilla, and precipitated by eating and drinking. A clinical examination had revealed no cause of the pain. Radiographs of the upper jaw showed a normal edentulous jaw in the molar region. No radiographs were apparently taken of the mandibular teeth, which were heavily restored. The patient was referred to an ENT Specialist who carried out an irrigation of the maxillary sinuses. Two weeks later she was still complaining of the pain and was seen at 22.00 hours one evening with an acute exacerbation of her pain. Both jaws were radiographed and massive residual caries was detected beneath a clinically sound and well-executed silver amalgam restoration in a mandibular premolar tooth. An interproximal cavity was detected in the first mandibular molar tooth, The premolar tooth was extracted and no further pain was exporienced.

Case 2 :- A 40 year old NCO was referred by the medical officer to an ENT Specialist with a probable diagnosis of trigeminal neuralgia involving the second division of trigeminal nerve on the left side. The pain, which was severe and paroxysmal, was precipitated by chewing and swallowing. It had been present for six weeks. A dental opinion several weeks previously had been that no cause could be found in the teeth. The otolaryngologist was anxious that a dental cause be excluded and requested a second opinion. Dental films were taken of all mandibular and maxillary teeth, including bite wing films. A large carious cavity involving the dental pulp horn was found on the posterior surface of the left maxillary second molar tooth below the gingival margin. The tooth was extracted and the patient had no further pain. (A careful clinical examination of the mouth revealed no sign of this cavity).

Dental caries does not provide the only means of exposure of the dental pulp, cementum or dentine of the tooth. Erosion, abrasion, attrition or tooth fracture can uncover sensitive tooth structure with subsequent pain but this is hardly likely to cause diagnostic difficulty. The presence of dissimilar metal restoration in immediate proximity to each other may, by galvanic action, produce pain e.g. a silver amalgam restoration next to a gold inlay.

Pulpitis may be caused by any mechanical, chemical or bacterial trauma and, although it is initially reversible, it may proceed to strangulation and suppuration. The pulp in this case strangulates just as the contents of a hernial sac strangulate. The pulp death can be very rapid with production of an acute alveolar abscess, or slow with production of a chronic apical abscess or years later with a dental cyst. Chronic apical abscessess may be associated with a mucosal, or rarely a cutaneous, sinus.

REFERRED PAIN

Referred pain is commonly encountered in dental practice. 'Head's areas' remain although not quite as Head described them. In general it can be said that in addition to feeling pain in the diseased tooth and the immediate area about it in the mouth, the patient may suffer referred pain in the skin of the face overlynig the tooth. The mandibular and maxillary third molar can give rise to referred pain in the ear, temporal region or neck. The mandibular first and second molars occasionally cause pain in the hyoid region or the ear. The mandibular premolars rarely give rise to pain in the hyoid region. It is commonly accepted that patients sometimes experience difficulty in locating the tooth causing pain and this should always be taken into account.

COMPLICATIONS OF TOOTH EXTRACTION

Alveolar osteitis or dry socket is an acute osteitis complicating exodontia. It is extremely painful and follows early loss of blood clot or failure of a clot to form, the socket wall then becoming infected by oral pathogens. The diagnosis is readily made; a history of recent extraction, foetor oris, severe pain in the area and a socket filled with dirty debris and slough instead of clean blood clot. The pain is out of all proportion to the lesion causing it.

PERICORONITIS

Inflammation of the follicle and soft tissues of the erupting tooth is termed pericoronitis (Kay, 1966). This condition is most commonly seen in young adults with an erupting third molar tooth but may be seen to a lesser degree with other erupting teeth. Pericoronitis may proceed to suppuration and be responsible in the case of the mandibular third molar tooth for deep neck infections. The condition is extremely painful and is accompanied by trismus, and foetor oris.

The mere presence of an unerupted, ectopic or impacted tooth does not imply that it is the cause of a patient's pain. Unless the tooth is itself the seat of some pathological process or is in commuuication with the mouth it is important that another cause for the patient's pain be sought.

CENTRAL LESIONS

Central lesions of the jaws (dental cysts, ameloblastoma, odontoma) do not cause pain unless they are infected or are pressing on or displacing a nerve. A metastatic carcinoma may cause pain and this could be its first presentation; although exeprience shows that those in the mandible usually cause paraesthesia of the lip.

Acute osteomyelitis of the jaw is a most painful condition and the combination of history of trauma or a dental abscess, with anaesthesia of the lip, mobile teeth and severe pain suggest this surgical disaster. Acute maxillary osteomyelitis is a rare disease and is usually seen in the infant.

Periostitis associated with contiguous soft–tissue infection produces a variable amount of subperiosteal new bone formation. It is especially noticeable in the growing child, to the extent that neoplasm is suspected. The condition is painful and a radiograph of the infected jaw shows new bone formation.

Jaw fractures are usually readily diagnosed, but are not always painful, and a careful examination will need to be made.

Necrotic and ulcerative conditions of the mouth, whether infective or neoplastic, can be very painful, but rarely provide any diagnostic problem as a cause of facial pain. Vincent's disease or trench mouth, is not uncommon in the adolescnt and young adult or debilitated patient. In the young infant's mouth the herpes simplex virus causes a gingivo-stomatitis which clinically mimics Vincent's infection. The aphthous ulcer, with its yellow halo and red centre, is extremely common in its minor form and readily recognizable. The vast spectrum of treatments recommended suggest that none is really efficacious.

MANDIBULAR JOINT

The mandibular joint is a pain–sensitive structure and may suffer trauma of an acute or chronic nature. A blow to the jaw insufficient to produce condylar fracture will cause an effusion with severe local pain and limited movement for a short period. Arthritic changes are occasionally a part of a generalized condition but are commonly due to some intra-oral cause : malocclusion, loss of teeth or loss of vertical dimension (mandibular overclosure) or poorly executed dental restoration. It would be safe to say that most patients with mandibular joint dysfunction have an intraoral cause. The patient, almost invariably female, presents with one or more symptoms. These include pain, in or about the mandibular joint and referred to the temple, ear, tongue, mandible or neck. Limited jaw movements, early morning trismus, joint crepitus, recurrent subluxation, and very rarely, tinnitus also occur.

SINUS DISEASE

Finally it is pointed out that toothache is not always due to dental abnormality, and it is worth while stressing that maxillary sinusitis can cause pain in the maxillary teeth. Usually all the posterior maxillary teeth on the involved side are tender to percussion but this should not be relied upon. Some patients present with pain in one tooth only. It is most often the dentist who needs to make this differential diagnosis.

and who finds to his cost that it required some determination to resist the request for extraction by a patient who is convinced that a particular tooth is the cause of his pain.

Case 3. – A 30 year–old civilian patient with a good dentition but a large number of well–executed restorations was referred by his medical practitioner for an opinion as to the cause of his maxillary pain. He had an upper respiratory – tract infection ten days previously. He was adamant that the maxillary first molar tooth was the cause of his pain and was most insistent that it be extracted. All the left maxillary posterior teeth were tender to percussion and in addition the alveolar mucosa was tender to palpation. Dental X–Ray and pulp tests demonstrated no abnormality. Transillumination of the sinuses was inconclusive. It was felt, however, that maxillary sinusitis was the possible cause of (this man's) pain and he was referred to an ENT specialist who confirmed the diagnosis and treated him successfully.

OTHER CAUSES

Aerodontalgia is rare and peculiar form of dental pain encountered in those who fly. The change in altitude causes the pain in restored or non–vital teeth because, it is thought, trapped gases expand inside the pulp canal.

For many years the presence of small calcareous masses in the dental pulp, known as pulp stones, were thought to be a cause of pain. There is no evidence to support this thesis and pulp stones are seen in dental radiographs of symptomless teeth. They should never be offered as a cause for the patient's pain although in the past numerous teeth have doubtless been extracted as a penalty for possessing pulp stones.

A condition known as 'menstrual toothache' is described in the dental literature. It is possible that the pain is due to hyperaemia of the dental pulp.

SUPERIOR CONSTRICTOR MUSCLE

The lingual flange of an over–extended mandibular denture may bear on the superior constrictor muscle, producing dysphagia, pain in the throat, floor of the mouth and upper part of the neck. Adjustment of the denture produces a cure. Ill–fitting dental appliances are a potent cause of discomfort and pain and a dental opinion should always be obtained; the temptation to treat the patient with topical applications should be resisted.

CONCLUSION

The patient with pain in the face or jaws be seen by specialists from several disciplines, or by general practitioners in medicine or dentistry. Prudence dictates that since dental abnormality is so common an active effort be made to exclude it before the other less common causes of facial pain are diagnosed. Dental pain arising from carious y mimic trigeminal neuralgia. It behoves us to remember this when igeminal neuralgia, especially in the younger patient. A patient with his (or even an edentulous patient) is more likely to have dental disorder than al neuralgia or demyelination. It should not be forgotten that dental carries is ommonest disease affecting man.

SUMMARY

This article sets out to stress what is all too often forgotten : that dental caries is the commonest cause of facial pain. It is frequently not obvious and a full clinical examination of the teeth, together with peri-apical and bite wing radiographs and pulp tests of the teeth are required to detect it and its pulpal sequelae.

Other dental causes have been discussed.

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